JUL 3 1930

Vol. V JUNE, 1930 No. 5

THE AMERICAN HEART JOURNAL

©Am. Ht. Assn.

ADVISORY EDITORIAL BOARD

HENRY A. CHRISTIAN
ALFRED E. COHN
LEROY CRUMMER
ELLIOTT C. CUTLER
GEORGE DOCK
JOSIAH N. HALL
WALTER W. HAMBURGER
JAMES B. HERRICK
E. LIBMAN
WM. McKIM MARRIOTT
JONATHAN MEAKINS
JOHN H. MUSSER
JOHN ALLEN OILLE
STEWART R. ROBERTS
G. CANBY ROBINSON
LEONARD G. ROWNTREE
ELSWORTH S. SMITH
WM. S. THAYER
PAUL D. WHITE
CARL J. WIGGERS
FRANK N. WILSON

PUBLISHED BI-MONTHLY
UNDER THE EDITORIAL DIRECTION OF
THE AMERICAN HEART ASSOCIATION

LEWIS A. CONNER - - - - - - - Editor
HUGH McCULLOCH - - Associate Editor
EVELYN HOLT - - - - Associate Editor

PUBLISHED BY THE C. V. MOSBY COMPANY, 3523-25 PINE BLVD., ST. LOUIS, U. S. A.
Copyright, 1930, by The C. V. Mosby Company
Entered at the Post Office at St. Louis, Mo., as Second Class Matter.

The American Heart Journal

CONTENTS FOR JUNE, 1930

Original Communications

Reminiscences of the Late Professor Willem Einthoven. By A. Samojloff, Kasan, U. S. S. R. ---- 545

The Effect of the Intravenous Administration of Digitalis in Paroxysmal Tachycardia of Supraventricular Origin. By Frank N. Wilson, M.D., and Shelby W. Wishart, M.D., Ann Arbor, Mich. ---- 549

The Blood Vessels as a Possible Source of Visceral Pain. By W. K. Livingston, M.D., Portland, Oregon ---- 559

The Relationship of Auriculo-ventricular Conduction Time in Rheumatic Fever to Salicylate Therapy. By John Wyckoff, M.D., Arthur C. DeGraff, M.D., and Solomon Parent, M.D., New York, N. Y. ---- 568

The Visualized Esophagus in the Differentiation of Lesions of the Right and Left Heart. By Leo G. Rigler, M.D., Minneapolis, Minn. ---- 574

The "Latency Theory" of Heart-Block and Interpolated Ventricular Premature Beats. By Richard Ashman, Ph.D., New Orleans, La. ---- 581

Diphtheria as a Cause of Late Heart-Block. By Stuyvesant Butler, M.D., and Samuel A. Levine, M.D., Boston, Mass. ---- 592

The Distribution of the Potential Differences Produced by the Heart Beat Within the Body and at Its Surface. By Frank N. Wilson, M.D., Ann Arbor, Mich. ---- 599

Partial Bundle-Branch Block. A Case of Three-to-One and Four-to-One Block. By Solomon R. Slater, M.D., Brooklyn, N. Y. ---- 617

The Local and Systemic Effects of Arterio-venous Fistula on the Circulation in Man. By Laurence B. Ellis, M.D., and Soma Weiss, M.D., Boston, Mass. ---- 635

Cardiac Symptoms Not Due to Cardiac Disease. By Henry Farnum Stoll, M.D., Hartford, Conn. ---- 648

Heart Disease in the State of New York. A Statistical Review of Mortality and Morbidity. By J. V. DePorte, Ph.D., Albany, N. Y. ---- 652

The Cardiac Clinics of New York; Their Origin, Aims and Accomplishment. By Edwin P. Maynard, M.D., Brooklyn, N. Y. ---- 660

Department of Clinical Reports

Complete Heart-Block of Seven Years' Duration in a Child Resulting from Injury. By T. Homer Coffen, M.D., Portland, Ore. ---- 667

Insulin Shock as the Cause of Cardiac Pain. By Kenneth B. Turner, M.D., New York, N. Y. ---- 671

Department of Reviews and Abstracts

Selected Abstracts ---- 673

The American Heart Journal

Vol. V | June, 1930 | No. 5

Original Communications

REMINISCENCES OF THE LATE PROFESSOR WILLEM EINTHOVEN*

A. Samojloff
Kasan, U.S.S.R.

DR. WHITE asked me if I would tell of my reminiscences of the late Professor Einthoven. I knew, esteemed, and loved this man, and therefore I willingly accepted the invitation. I only regret that insufficient command of English does not allow me to express clearly and plainly all my thoughts and feelings. As you all know, Einthoven worked almost exclusively in the region of electrophysiology. This branch of physiology stood for a long time completely isolated from life, medicine, and even from the general path of the development of physiological knowledge; shut off in this way, electrophysiology could not progress and it seemed that it would be difficult to alter this sad situation of the study of animal electricity.

Among different favorable circumstances I will point out the one which opened at once new possibilities for electrophysiology, namely, the invention by Lippman, a physicist in Paris, of a new instrument, the capillary electrometer. This instrument rapidly proved its usefulness in the study of electrical phenomena in the animal body, especially after Waller showed that the capillary electrometer was able to register not only the action currents of an isolated animal heart, but also the action currents of the heart of an intact animal and even the action currents of the human heart.

In my youth I became interested in electrophysiology, and when I saw for the first time in a microscope the movements of the mercury meniscus produced by the heart, I may say I was conquered by electrophysiology forever. With the modest instruments I had at the time, I tried to record the curves of a capillary electrometer, and I was thrilled when successful.

One day I obtained a copy of Einthoven's article in Pflüger's *Archives* with records of the action currents of a human heart: I

*Presented at the Massachusetts General Hospital, Boston, September, 1929.

understood that my technique was only a feeble attempt at the solution of the problem which in the hands of Einthoven was brought to the state of complete perfection. I spent hours examining these curves, and asked myself: Who is this man, what is he like and by what means does he obtain such results? Such sharpness distinguishing the curves of Einthoven was never attained before. But such curves soon ceased to satisfy Einthoven on account of their inability to give accurately the changes of action with the time. He developed a method of correcting the curves and then I admired not only his technique but also his theoretical knowledge. These two accomplishments showed the essential features of Einthoven's talent. His mind worked like an instrument of precision. He worked only on what could be measured, and his measurements reached the limit of precision possible under the circumstances. Later we all could see that the subjects he chose for his investigations always lent themselves beautifully to precise measurements.

I met Einthoven for the first time a few days before the opening of the Physiological Congress in Brussels in 1904. I wrote to him from Paris where I was at the time, and then I stopped at Leyden to visit him. I saw at the same time Einthoven and his new instrument, the string galvanometer, which later became known the world over. Einthoven had just completed the construction and installation of his instrument. He was enthusiastic over his extraordinary success, with visions of the possibilities that were open. What I saw there seemed to me unattainable.

After inspecting the laboratory we went together for a walk in the suburbs of Leyden. With great pleasure I recall our long discussions of the importance and the future of electrophysiology. I was glad to find in him support of my idea that electrophysiology must not only go further in studying the nature of animal electricity, but that, first of all, it must enter as a new method of investigation into all branches of physiology. Every organ of the body, so far as it represents a collection of excitable elements, must be studied by the electrophysiological method, because every excitable tissue functions as a source of electricity at the moment of the excitation.

Beginning with the first meeting with Einthoven our correspondence started and continued till the end of his life. I shall speak only briefly of his scientific merits, for they are still too fresh in everybody's mind.

First of all he is the creator of electrocardiography. All methods proposed by him, his standardization, his electrodes, his three leads, his triangle, his terminology, all these are based on theory and at the same time are highly practical; they will all remain in electrophysiology, if not forever, at least for a long time.

Later Einthoven demonstrated how to experiment with the currents

of nerve fibers. He was the first to demonstrate the action current produced by heart contractions in the depressor nerve in its centripetal nerve fibers. He was the first to demonstrate the action current in the sympathetic nerves.

Later he did not adopt the method of amplification with vacuum tubes. He told me several times that by perfecting his string galvanometer he would obtain more correct curves than with vacuum tubes. During the last years of his life he was working with strings $1/100$ of a micron thick. Such strings are so thin that they show Brownian movement due to the bombardment of air molecules. The weight of such strings is so small that they could show all the vibrations produced by a voice. They vibrate together with the surrounding air. Photographic records of such strings in air were the best and simplest method of recording sound waves. Einthoven obtained beautiful curves of vowels, for example. The strings were so thin that the question rose as to how they could be seen and projected; Einthoven wrote a special paper on this question.

Einthoven did not write much. All his works are distinguished by thoroughness, completeness and particular beauty of execution.

It happened that I used to meet Einthoven comparatively often. Due to certain circumstances I used to stay with his family, in his house, for several weeks, and could observe his life. His was a pleasant life. He experienced real happiness achievable only by great investigators who tread new scientific paths. He was happy in his family life, surrounded by the love and kindness of his family and friends. He was given the greatest scientific honors and signs of distinction, including the Nobel Prize. But he remained the same simple, frank, direct man. His definiteness and frankness, seen in his work, could be discerned also in the features of his character. His frankness compelled him to fight all he considered unjust, and on the other hand this same frankness made him welcome all who were worthy of his support.

One of the features of his character, which I myself also possess, closely united us, that is, the love to joke. He liked jokes, liked to laugh, liked also to make friendly fun of people and did not object to being made fun of himself. Often I could observe that a joke would lead him to a remark revealing his noble side. A few years ago I was glancing through Pflüger's *Archives* and noticed that the paper in which, for the first time electrocardiograms of a string galvanometer were printed, would soon be 25 years old. I sent Einthoven on this occasion a joking letter. I wrote him: "Dear Einthoven: I am writing this letter not to you, but to your dear and honorable string galvanometer, and therefore I address him. Dear, honorable galvanometer, I just learned that you have a jubilee, that 25 years ago you traced the first electrocardiogram. I congratulate you. I do not

want to keep away from you the fact that I am fond of you in spite of your being sometimes very tricky. I marvel at how much you have accomplished during 25 years. If we should count the number of meters and kilometers of photographic paper used for records with your strings in all parts of the world, the resulting figures would be enormous. You created a new industry, you have philological merits too; we owe you the birth of new words like electrocardiogram."

At the end of this long letter which I do not want to reproduce in full, I said: "And so, my dear and honorable galvanometer, I embrace you and beg you to transmit to Einthoven my congratulations." Then I added: "Dear Einthoven, I beg you to read this letter to the string galvanometer, since it can write but it can't read."

In a short time I received an answer from Einthoven; he wrote: "I have carried out precisely your request and read to the galvanometer your letter. Apparently he listened and took in with pleasure and joy, all that you wrote. He hadn't suspected that he had done so much for humanity, but at the place where you said he does not know how to read, he all of a sudden became furious, so that I and my family became even alarmed. He cried: 'What, I can't read? It's a terrible lie. Do I not read all the secrets of the heart?' I calmed him and advised him in the future to continue to do only one thing—to work and to toil as much as he could for the benefit of humanity, and not to think of gratitude."

This idea of work for humanity I saw in Einthoven very often in different ways. When in Stockholm in 1924 he received the Nobel Prize he ended his speech with the following words: "Es ist ein neues Kapitel der Lehre der Herzkrankheiten hinzugefugt worden nicht durch die Arbeit eines einzigen, sondern durch diejenigen vieler talentvoller Männer, die bei ihren Forschungen sich durch keine politischen Grenzen haben beeinflussen lassen und, über unsere Erdoberfläche verbreitet, Ihre Krafte einem idealen Zwecke haben widmen wollen der Entwickelung der Wissenschaft, wodurch schliesslich der leidenden Menschheit genützt worden ist."*

A few months before his death in 1927, when nobody was suspecting anything out of the way I met him in Leyden. He was gay and brisk as usual. He gave me as a souvenir an excellent photograph of himself. At the station when we were parting, suddenly he became serious: "Perhaps we may never meet again." He proved to be right.

In memory of today's talk may I ask to be allowed to send after I reach home a copy of this picture of Einthoven to the cardiac department of the Massachusetts General Hospital?

*A new chapter in the scientific knowledge of heart disease has been introduced, not through the work of a single person, but through the labor of many talented men who have carried out their investigations unlimited by any political boundaries. These individuals over the whole world have dedicated their energy to an ideal consisting of the development of knowledge which ultimately benefits suffering mankind.

THE EFFECT OF THE INTRAVENOUS ADMINISTRATION OF DIGITALIS IN PAROXYSMAL TACHYCARDIA OF SUPRAVENTRICULAR ORIGIN*†

FRANK N. WILSON, M.D., AND SHELBY W. WISHART, M.D.
ANN ARBOR, MICH.

IN THE great majority of instances simple paroxysmal tachycardia occurs in attacks, which, if they do not terminate spontaneously within a few hours, can be brought abruptly to an end by pressure upon the carotid sheath or by some other simple procedure involving vagus stimulation. Consequently, there is ordinarily little need for the administration of drugs in this disorder, and comparatively few careful studies of the effects of medicinal therapy have been made.

There is considerable evidence that both digitalis and quinine, or quinidine, are not infrequently effective in restoring the normal cardiac mechanism. When the administration of these or other drugs by mouth is followed after a brief interval by the return of sinus rhythm, it is, however, difficult, unless this effect is obtained repeatedly, to exclude the possibility that the attack may have ended spontaneously rather than as a result of the treatment. When on the other hand, the paroxysm ends abruptly immediately after the intravenous injection of a drug, there can be little doubt as to its effectiveness.

Singer and Winterberg[2] have reported a number of instances in which the intravenous administration of quinine (0.4 to 0.75 gm.) was followed at once or within a few minutes by cessation of the abnormal cardiac mechanism. There was usually a striking fall in heart rate preceding the termination of the paroxysm. Iliescu and Sebastiani[3] also have reported an instance in which quinidine by mouth strikingly reduced the rate of the ectopic rhythm. It has also been observed that strophanthine by vein or digitalis by mouth will occasionally abolish the abnormal rhythm; there is, however, so far as we know, no clear evidence that digitalis preparations given intravenously may produce pronounced slowing of the heart rate followed by cessation of the paroxysm, just as in the case of quinine. We wish therefore to report two cases in which these effects were observed.

OBSERVATIONS

An American housewife (E. T.), aged 54 years, was admitted to the hospital on March 3, 1925, complaining of attacks of rapid heart action which began after an attack of rheumatic fever at the age of eighteen years. The attacks occurred every two to four months and varied in duration from fifteen minutes to thirty hours. They were accompanied by palpitation and giddiness and were followed by

*From the Department of Internal Medicine, University of Michigan Medical School.
†The observations reported here were referred to briefly in an article read at a meeting of the Association of American Physicians, May 1926. (1).

severe nausea and vomiting. Cardiovascular examination showed moderate enlargement of the heart and low-grade aortic insufficiency. The peripheral vessels were moderately sclerosed, the blood pressure was 145 mm. Hg. systolic, 65 mm. Hg. diastolic, and the electrocardiogram showed pronounced left ventricular preponderance. The thyroid gland was moderately enlarged and there were rather questionable signs of mild thyroid intoxication. The cardiac function was good.

The cardiac mechanism was normal until March 11, when the heart rate suddenly rose to 200 or more per minute and the symptoms described by the patient as characteristic of previous attacks developed. A summary of the observations made at this time is given in Table I. The rates given in the table were computed from

Fig. 1.—First patient; E. T. All curves taken by a chest lead, manubrium to ensiform. Ords. 1 cm. equals 1 millivolt. Abs. 1 div. equals 0.04 sec. 3797; control; rate 205 per minute. 3798; after 0.2 gm. quinidine; rate 210; QRS interval slightly increased. 3799; after digifolin 10 c.c. intravenously; rate 174. 3800-1; after second dose of digitalis; normal rhythm. 3800-2; 2-to-1 heart-block during an attack of nausea; taken immediately after 3800-1.

electrocardiograms which were taken at brief intervals throughout the duration of the attack. In order to avoid the muscle tremor which distorted the curves taken in the usual manner a chest lead (manubrium to ensiform) was employed. The form of the ventricular complex indicated that the abnormal rhythm was of supraventricular origin, but no auricular deflections were visible. Bilateral pressure upon the carotid sheaths and ocular pressure were without effect. The intravenous administration of quinidine (0.2 gm.) had no effect upon the heart rate, but produced minor changes in the form of the ventricular complex and increased the

QRS interval some 0.03 or 0.04 second (Fig. 1). A proprietary preparation of digitalis (10 c.c. of this preparation are approximately equivalent to 1 mg. of ouabain) was then given intravenously. An immediate and striking fall in heart rate resulted. A half-hour later a second dose (5 c.c.) was given; at this time the movements of the galvanometer string were under observation. There was a further slowing of the rate followed immediately by cessation of the abnormal rhythm. Nausea and vomiting followed and during the attacks of vomiting two-to-one heart-block was present (Fig. 1). The nausea continued for 48 hours, diminishing gradually. The patient had no further attacks of tachycardia while she remained in the hospital.

The second patient, E. M., a factory worker, aged 32 years, was admitted to the hospital Jan. 5, 1926. He was suffering from an attack of rapid heart action, associated with palpitation, dyspnea, and exhaustion, which began 12 days before. He had his first attack while playing baseball at the age of 14 years. At first the attacks came only twice a year and lasted only a few hours; but for three years he had had from six to nine attacks annually, each lasting from eight to twelve days. The attacks usually came on during exertion, but occasionally they began while he was walking or sitting down, or awakened him from sleep. They were accompanied by severe prostration and dyspnea and he was forced to go to bed; in the longer attacks he suffered from insomnia and profuse sweating at night, and, after four or five days, from cough with thick tenacious sputum. There was a clear history of rheumatic fever five years before admission.

TABLE I

E. T.—PAROXYSMAL AURICULAR TACHYCARDIA

E.C.G. NO.	DATE	TIME	MEDICATION	TIME SINCE LAST DOSE	HEART RATE	REMARKS
3794	3/11/25	12:30 P.M.			211	No digitalis since 3/3/25
3795	3/11/25	12:31 P.M.			208	During bilateral vagus pressure
3796	3/11/25	12:32 P.M.			206	During ocular pressure
3797	3/11/25	12:45 P.M.			205	Control
3797	3/11/25	12:46 P.M.	Quinidine grs. iii 1. V.		208	Taken during injection of quinidine
					208	Taken after injection of quinidine
3798	3/11/25				206	Last curve eight minutes after
				8 min.	210	
	3/11/25	1:18 P.M.	Digifolin 10 c.c. 1. V.			
		1:25 P.M.		7 min.	176	
3799	3/11/25	1:26 P.M.		8 min.	173	
		1:27 P.M.		9 min.	174	
3800	3/11/25	1:47 P.M.	Digifolin 5 c.c. 1. V.		107	Further slowing followed immediately by normal rhythm. Nausea and vomiting with partial heart-block during attacks of vomiting.

The heart rate was approximately 208 per minute; the precordium and the engorged neck veins throbbed violently. The patient was obviously short of breath and there was slight edema of the sacrum, a slight hydrothorax on the left side, râles at the lung bases, engorgement of the liver, and a small collection of ascitic

fluid. The heart was considerably enlarged to the left (left border 3 cm. outside the nipple line and 16 cm. to the left of the midline); embryocardia was present, but there were no murmurs. The urine contained a small amount of albumin and there was a slight leucocytosis. The electrocardiogram showed small ventricular complexes with broad initial deflections; no auricular deflections were visible.

TABLE II

E. M.—PAROXYSMAL AURICULAR TACHYCARDIA

E.C.G. NO.	DATE	TIME	MEDICATION	TIME SINCE LAST DOSE	HEART RATE	REMARKS
5400	1/6/26	11:15 A.M.			211	Pressure on both vagi without effect
5401	1/6/26	11:30 A.M.			210	Control
	1/6/26	11:31 A.M.	Philocarpine ⅛ gr. 1. V.			
5401	1/6/26				200 to 210	Taken shortly after injection of pilocarpine
5402	1/6/26	11:45 A.M.			200 to 204	During ocular and vagal pressure
5403	1/6/26	12:06			204	Control
	1/6/26	12:07	Apomorphine Grs. 1/10 by hypo.			
5403	1/6/26	12:28			204	
5411	1/7/26	1:05 P.M.			210	Control
	1/7/26	1:07 P.M.	Digifolin 10 c.c. 1. V.			
5412	1/7/26	1:17 P.M.		10 min.	190	
5413	1/7/26	1:30 P.M.		23 min.	187	
5414	1/7/26	2:00 P.M.		54 min.	189	
	1/7/26	2:15 P.M.	Digifolin 5 c.c. 1. V.			
5415	1/7/26	2:25 P.M.		10 min.	182	
5416	1/7/26	2:50 P.M.		35 min.	185	Change in form of ventricular complexes
	1/7/26	3:40 P.M.		85 min.		Normal rhythm reported
5421	1/7/26	4:25 P.M.		130 min.	88	Normal rhythm

A summary of effects of various procedures upon the abnormal rhythm is given in Table II. While each of these procedures was carried out the galvanometer string was watched, and records were made at frequent intervals. The rates given are computed from these records. Ocular and vagal pressure were without effect. Pilocarpine intravenously and apomorphine hypodermatically, which induced vomiting, also failed to abolish the abnormal rhythm or to modify the heart rate. As in our first patient intravenous administration of a proprietary preparation of digitalis was followed immediately by a striking drop in the heart rate (Fig. 2) and a further fall in rate followed a second smaller dose. Thirty-five minutes after the second dose the ventricular complexes changed in form, and assumed the shape of the complexes recorded after the normal rhythm returned (Fig. 3). It seems clear that the broad initial deflections of the complexes recorded earlier was the result of defective intraventricular conduction dependent upon the rapid rate, and that when the rate fell the conducting system recovered its normal conductivity. Recovery did not take place at once for the rate was actually slightly higher when the change in the form of the complexes was observed than it was twenty-five minutes earlier. It seems possible, therfore, that there was some tendency to cumulative fatigue of the intraventricular conducting system and that the immediate previous history of these tissues and not merely the length of the preceding diastole was a

factor in determining intraventricular conductivity. Although the return of the normal rhythm was somewhat delayed, there can be little doubt that it was the result of the treatment. We have observed that when digitalis or ouabain is given intravenously in auricular fibrillation there is an immediate drop in the ventricular rate, but the maximum effect does not appear as a rule until two or three hours after the drug is given. Following the return of normal rhythm the heart gradually decreased in size and there was a pronounced diuresis with a loss of 31 lbs. in weight. Beginning on January 9 the patient received 4 c.c. of the standard tincture of digitalis daily for a period of eight days without developing toxic symptoms. He had no further attacks of tachycardia while under observation; an electrocardiogram taken on Jan. 15 showed occasional ventricular extrasystoles with retrograde stimulation of the auricles; the ventricular complexes were not remarkable except that the T-deflections were inverted probably as a result of the administration of digitalis.

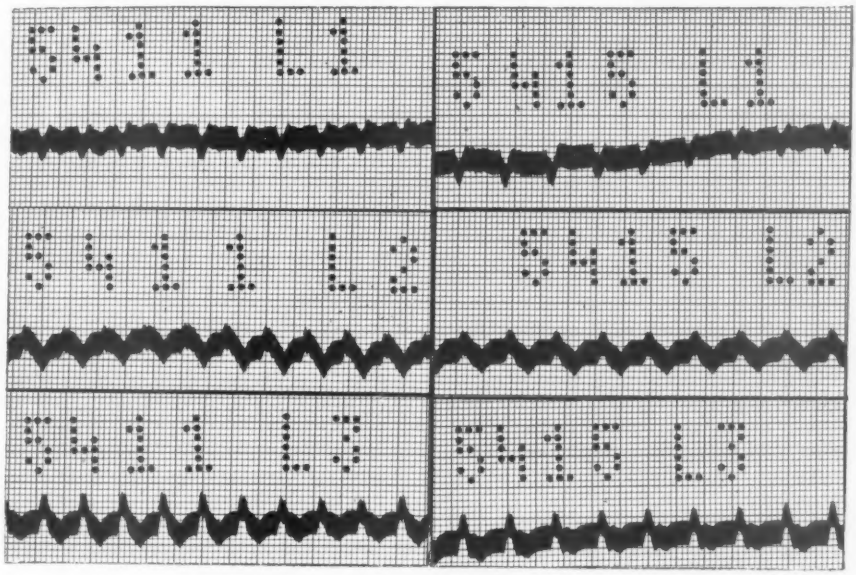


Fig. 2.—Second patient; E. M. The three standard leads. 5411; control; rate 210 per minute. The ventricular complexes are small; the initial deflections, broad. 5415; 10 minutes after digifolin 10 c.c. intravenously; rate 182.

DISCUSSION

The mechanism by which digitalis slowed the rate of the ectopic rhythm and finally abolished this rhythm in our two patients is obscure and must remain so until the nature of paroxysmal tachycardia is better understood. The effects of digitalis upon the heart are complex and they have not been completely investigated. The increase in vagal tone which it induces tends to diminish the rate of stimulus production within the sinus node and within the atrio-ventricular node, and to depress the conductivity of the junctional tissues. It reduces the refractory period of the auricular muscle, and thus improves intra-auricular conduction when the rate of beating is so rapid that the excitatory process is advancing through partially refractory tissue.[4]

By its direct action upon the cardiac tissues, as opposed to its indirect effects exerted through the vagus, digitalis lengthens the refractory period of the auricular muscle,[4] depresses the conductivity of the junctional tissues, and enhances the rate of stimulus formation in those centers which lie below the auriculo-nodal junction.

The first question that arises in the study of any digitalis effect is whether this effect is due to a direct action of the drug upon the heart muscle or to an increase in vagal tone. This question can be answered by the administration of adequate doses of atropine, but in the present instance this procedure was not carried out because the condition of

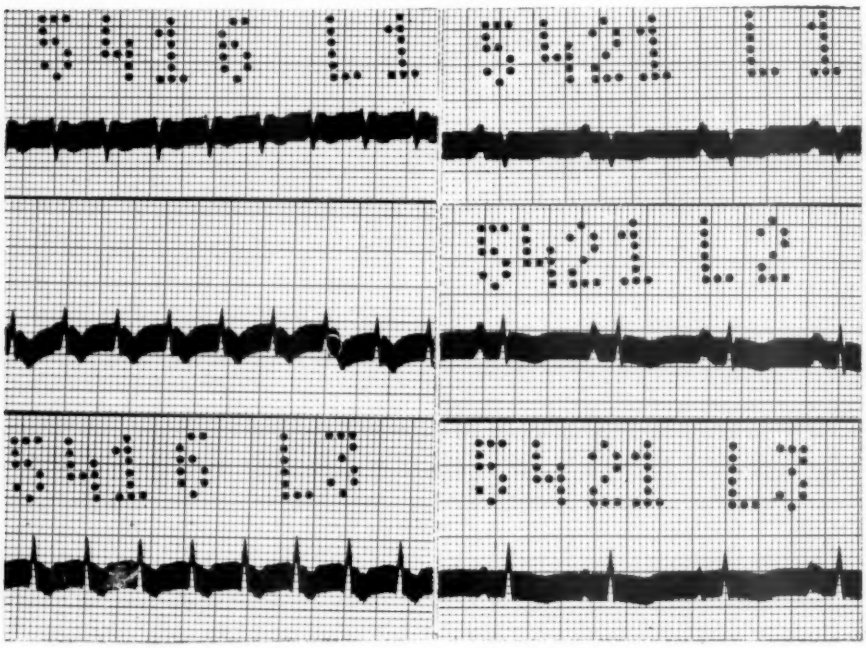


Fig. 3.—Second patient; E. M. The three standard leads. 5416; the ventricular complexes have now assumed the form which they showed after normal rhythm returned; rate 185. 5421; normal rhythm rate 88.

our patients made any medication which might tend to prolong the abnormal rhythm inadvisable. The fact that pressure upon the carotid sheath and upon the eyeballs was without effect upon the ectopic rhythm in both patients, and that pilocarpine, which stimulates the vagal endings, and apomorphine which stimulates the medullary centers, were without effect in one of them does not necessarily mean that the digitalis effects under discussion were non-vagal in origin. The failure of these procedures in some cases of paroxysmal tachycardia and their success in others is probably due in greater measure to differences in the grade of vagal stimulation which they induce than to variations in the susceptibility of the abnormal cardiac mechanism to

vagal influence. The effects of these methods of stimulating the vagus are equally variable when sinus rhythm prevails.

Opposed, however, to the view that digitalis slowed the rate of the paroxysmal rhythm in our patients by its vagal rather than by its direct effects upon the cardiac muscle is the fact that pressure upon the carotid sheath and other methods of stimulating the vagus do not ordinarily slow the rate of the ectopic rhythm in paroxysmal tachycardia even when they abolish the abnormal mechanism. Occasionally, however, there is very slight slowing involving only two or three cycles, just at the end of the paroxysm.[5] Exercise which reduces vagal tone is also usually without effect although Wenckebach and Winterberg[6] mention cases in which an increase in rate was observed following exertion.

In view of these considerations it is impossible to decide whether the effects produced by digitalis in paroxysmal tachycardia are vagal or

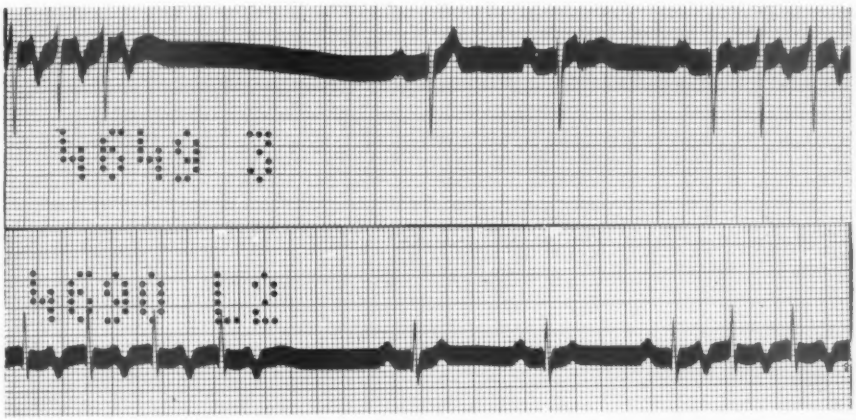


Fig. 4.—Curves from a case paroxysmal tachycardia of auricular origin. 4649; chest lead, manubrium to ensiform. An attack of paroxysmal tachycardia brought to an end by pressure upon both eyeballs. The attack ends with a blocked auricular beat. At the end of the record a new attack begins. 4690; Same patient, lead II. An attack of paroxysmal tachycardia ends spontaneously with a blocked auricular beat. At the end of the record a new attack begins.

non-vagal in origin until it is known whether or not they are altered by the administration of atropine.

The failure of quinidine to abolish the abnormal rhythm or to slow its rate in one of our patients raises the question as to whether the cardiac mechanism in cases in which digitalis is effective differs from that present in cases in which quinidine or quinine is effective. It is possible that the dose given was too small to produce the characteristic effects of the drug. Previous experience with the intravenous administration of quinidine made us hesitate to give a larger amount because of the marked depression of intraventricular conductivity which results when the ventricular rate is greatly accelerated. The dose given is sufficient to reduce conspicuously the rate of the circus rhythm in auricular flutter and in auricular fibrillation although the fall in rate

is more or less proportional to the original rate. Thus, in four cases of auricular flutter with original rates of 360, 291, 270, and 210 the reduction in rate produced by 0.2 gm. of quinidine given intravenously amounted to 75, 55, 52, and 23 beats per minute, respectively. In auricular fibrillation the fall in circus rate is usually 100 cycles per minute or more.

The observations recorded in this article were prompted by the study of another patient with paroxysmal tachycardia of supraventricular origin, but without visible auricular waves in the electrocardiogram. In this instance 0.5 gm. of quinine intravenously reduced the heart rate from 250 to 204 per minute within a period of two minutes; the abnormal rhythm was not, however, abolished. A digitalis preparation (digifolin 5 c.c.) was then given intravenously. The attack came to an end about one hour later, but unfortunately no electrocardiograms were made between the time when the digitalis was given and the end of the attack. These observations lead us to suspect that the failure of quinidine and the effectiveness of digitalis in the first case described in this article was probably not due to any peculiarity of the underlying mechanism, and suggest that the two drugs may possibly be given together with advantage in stubborn cases of paroxysmal tachycardia.

Because of the similarity of the effect of quinidine and quinine upon the heart rate in paroxysmal tachycardia to their effect upon the rate of the circus rhythm of auricular flutter and auricular fibrillation, it has been suggested that paroxysmal tachycardia is also due to circus contraction.[3] The effect of digitalis upon the rate of the ectopic rhythm in paroxysmal tachycardia is, on the other hand, just the reverse of its effect upon the circus rate in auricular flutter and auricular fibrillation, for although the change in the rate of the circus rhythm which it produces in these conditions is as a rule not profound, nevertheless there is a definite tendency for the rate to go up, and flutter can usually be converted into fibrillation by full digitalization. It is true that in rare instances of fibrillation, possibly because the direct effect of digitalis upon the auricular muscle overbalances its vagal effect, slight slowing of the circus rate results,[4] but this effect has not been observed in flutter, in which the gap of responsive tissue is wider than in fibrillation, and it does not seem to offer a reasonable explanation of the effect of the drug in paroxysmal tachycardia.

It has also been suggested that quinidine and its allies slow the heart rate in paroxysmal tachycardia and abolish the ectopic rhythm by lengthening the refractory period and thus rendering very early responses impossible.[7] It is conceivable also that the depression of excitability produced by these drugs might in some manner account for the effects in question. Digitalis, however, although its vagal and its direct effect upon the refractory period are in opposite directions, tends on

the whole to shorten the refractory period of the auricle in man; upon excitability it has no known effect.

In so far therefore as the effects of digitalis and quinidine upon the *auricular muscle* are known, there are apparently no similarities between them which can explain why they should act alike in paroxysmal tachycardia of auricular origin. It must be admitted, however, that the observations here reported do not demonstrate that these two drugs do have the same effect in this condition, for the effect of quinidine was not tested in one of the patients studied, and it was not adequately tested in the other, and the observations upon the third patient mentioned although suggestive were inconclusive. It was, furthermore, impossible to determine in these patients whether the abnormal mechanism was of auricular or of atrio-ventricular origin. Finally, it must be pointed out that if, as we suspect, the two drugs are found when adequately tested to have the same effect in one and the same case, it must still be admitted that this effect may be produced by digitalis in one way and by quinidine in an entirely different way, or that the effect in question may be produced by a similar, but as yet unknown effect of both drugs upon the cardiac tissues.

Both quinidine and digitalis depress the conductivity of the atrioventricular tissues, although the effect of the former is somewhat diminished and under certain conditions entirely obscured by the reduction of vagal tone which it induces. The question arises therefore as to whether a similarity in the effects of these drugs upon the junctional tissues has any bearing upon their effect in paroxysmal tachycardia. It is quite possible that digitalis was effective in both of our patients, and it is conceivable that quinidine was ineffective in the one instance in which it was given, because the abnormal rhythm was of atrioventricular origin. In this connection we may call attention, however, to a feature of paroxysmal tachycardia which has been insufficiently emphasized; we refer to the great difficulty of inducing block while the abnormal rhythm is present. When one considers the pronounced tendency to heart-block that is present in auricular flutter, even when the auricular rate is only slightly above that ordinarily seen in paroxysmal tachycardia, the rarity with which heart-block is induced in the latter condition by pressure upon the carotid sheath, or upon the eyeball, or by the administration of digitalis is astonishing. It is still more so when one considers that the P-R interval in paroxysmal tachycardia is frequently considerably increased.

As an example of the difficulty of producing block in paroxysmal tachycardia we may cite the observations made upon our first patient. After the second dose of digitalis the paroxysm ended and nausea with temporary high grade partial heart-block occurred. It seems strange that heart-block did not occur while the rapid rhythm was present, although it is impossible to rule out the possibility that in this instance

the site of origin of the abnormal rhythm was in the junctional tissues below the region where digitalis and vagal block occur. It should be pointed out that vagal and digitalis block does occur in rare instances of paroxysmal tachycardia; such cases have been described by Wenckebach and Winterberg[6] and we have ourselves seen such a case. Nevertheless it is extremely rare. The explanation is obvious although its significance is not clear. The susceptibility of the ectopic rhythm to vagal stimulation is ordinarily so great that a degree of vagal stimulation insufficient to produce block brings the abnormal cardiac mechanism to an end. Not infrequently, however, when auricular paroxysmal tachycardia is abolished by vagal stimulation, the last auricular beat of the ectopic rhythm is blocked (Fig. 4); that is, the onset of block and the end of the paroxysm occur at the same instant. We cannot therefore dismiss the possibility that in some instances of paroxysmal tachycardia vagal stimulation brings the abnormal rhythm to an end by depressing the conductivity of the junctional tissues, nor can we deny the possibility that digitalis and quinidine may slow the abnormal rhythm and finally abolish it in the same way.

In view of the character of the evidence bearing upon this problem which is at present available, and in view of the great difficulty of harmonizing many observations with the possibility suggested above it does not seem profitable to pursue the subject further at this time. We bring the matter up in order that observations bearing upon it may be made as the opportunities arise. It is desirable first of all to know whether the effect of digitalis described is influenced by the administration of atropine; whether it occurs in paroxysmal tachycardia of atrioventricular origin only, or whether it occurs in all types of paroxysmal tachycardia of supraventricular origin; whether it is a rare or a common phenomenon; and whether quinidine and digitalis may both slow the rate of the ectopic rhythm in one and the same case.

SUMMARY

In two cases of paroxysmal tachycardia of supraventricular origin the intravenous administration of digitalis produced a pronounced slowing of the heart rate followed by the return of the normal sinus rhythm.

The significance of these observations is discussed.

REFERENCES

1. Wilson and Wishart: Trans. Assoc. Amer. Phys., **41**: 56, 1926.
2. Singer and Winterberg: Wien. Arch. f. inn. Med., **3**: 329, 1922.
3. Iliescu and Sebastiani: Heart, **10**: 230, 1923.
4. Lewis: The Mechanism and Graphic Registration of the Heart Beat. 3rd Ed. Shaw and Sons, London, 1925.
5. Wilson and Herrmann: Arch. Int. Med., **31**: 923, 1923.
6. Wenckebach and Winterberg: Unregelmässige Herztätigkeit. Engelmann, Leipzig, 1927.
7. Scott: Heart, **9**: 297, 1921-22.

THE BLOOD VESSELS AS A POSSIBLE SOURCE OF VISCERAL PAIN

W. K. Livingston, M.D.
Portland, Ore.

INTRODUCTION

EVEN before the advent of local anesthesia it had been noted that the abdominal viscera were insensitive to many forms of stimulation which caused severe pain when applied to somatic tissues. As operations under local anesthesia became more frequent it was shown that cutting, crushing and burning of the viscera failed to elicit pain reactions from the conscious patient. Again, the pain was not uncommonly experienced in areas at some distance from the diseased viscus and was frequently accompanied by vascular changes, hyperesthesia of the skin and muscles and muscular spasm in the body wall.

In the face of these observations was the obvious fact that visceral disease was often characterized by severe pain.

Two schools of thought arose to explain the apparent discrepancy between these observations. The first, whose foremost exponent has been Sir James Mackenzie[1] proceeded on the theory that there were no true pain pathways from the viscera to the higher centers. It was pointed out that the "referred" phenomena, expressing themselves in the somatic tissues of the body wall, always bore a definite relation to the diseased viscus, in that the same segment of the spinal cord supplied both the somatic area involved and the organ at fault. It was assumed that even in the absence of true pain pathways, impulses from the viscera might "overflow" into the common pain pathways from somatic tissues and thus give rise to sensations of pain that would be ascribed by the subject to the peripheral distribution of the particular pathways so involved. Disease of an internal organ, then, was silent or painful, depending upon whether or not afferent impulses from the organ overflowed into somatic pathways of specialized pain conduction. This theory seemed to be consistent with many clinical observations and explained in a beautiful manner the phenomena of referred pain, hyperesthesia and muscle spasm.

Although this theory was of considerable importance in calling attention to the value of pain analysis in diagnosis and did much to stimulate interest in visceral pain, certain objections to its full acceptance became evident. The accuracy with which a patient localized his pain as deep-seated in certain cases of gall-stone colic, distention of the bladder or renal pelvis, or the pain caused by a large bolus of food in the esophagus and many other clinical observations seemed to argue against the assumption that pain of visceral origin is of necessity expressed in somatic areas. Thus a second school of thought

arose, by which it is claimed that true visceral pain exists, and can be elicited by a proper ("adequate") stimulus.

The work of Hurst,[2] Ryle,[3] Payne and Poulton[4] and many other investigators has laid emphasis on abnormal degrees of tension in the walls of hollow viscera as constituting "adequate" stimuli for true visceral pain. It has been shown that under proper conditions, spasm of smooth muscle and increased intramural tension from abnormal distention, are each capable of giving rise to afferent impulses which are experienced as pain and ascribed by the subject with a fair degree of accuracy to the internal organs rather than to the somatic tissues of the body wall.

THE CONTROL OF BLOOD VESSEL CALIBER

The function of any organ must be dependent to a high degree on the caliber of its blood vessels. Although the smooth muscle of blood vessels has a tonus of its own and may be influenced by various metabolic substances in the circulation, such as adrenalin, the smooth and harmonious functioning of the vessels in various parts of the body must be dependent upon nervous control. The effector units of this control are a part of the "autonomic" nervous system and have been termed "vaso-motor" neurons. These neurons affect the degree of smooth muscle contraction, but must not be considered "motor" in the same sense that we apply this term to motor neurons supplying voluntary muscle. In case the motor neuron to skeletal muscle is divided, an immediate, flaccid paralysis results, followed later by atrophy of the muscle elements. Division of "vaso-motor" neurons, on the other hand, may be followed by temporary or lasting changes in the vessel caliber, but never results in a true paralysis nor is it followed by atrophy of the smooth muscle. Rather than considering these neurons as being "motor" we might look upon them as association units, subserving a harmonizing and coördinating function.

The blood vessels are thus seen to possess an autonomy of function very similar to that displayed by the gastro-intestinal tract after it has been deprived of its extrinsic nerves. This autonomy and the coördinating function of the extrinsic nerves to blood vessels is well illustrated by Cannon's[5] sympathectomized cats. These animals, from which Cannon had removed the entire peripheral portion of the sympathetic nervous system, maintained an apparently normal circulation under favorable environment, but were extremely sensitive to cold. Cannon attributed their sensitiveness to cold, in part at least, to the animals' inability to reduce the peripheral circulation in the normal manner.

The coördinating functon of the extrinsic nerves of the blood vessels must be dependent, not only on the autonomic neurons, but on an equally important system of afferent neurons from all parts of the body as well as from the walls of the vessels themselves. The afferent

side of the "Visceral Nervous System" has been but little studied. That it exists and that it serves an important function in various reflexes and body adjustments is indicated by the work of Carlson and Luckhardt,[6] and many other investigators. We now believe that there are visceral afferents from visceral tissues in all parts of the body, including the blood vessels. The actual demonstration of afferent fibers from vessels has lagged behind physiological evidence, but sufficient evidence of their existence has been afforded by study of degeneration changes following division of perivascular plexuses, and the demonstration of various types of receptor end-organs, including typical Pacini corpuscles, in the walls of blood vessels.

The similarity in nervous control of the smooth muscle of blood vessels and the smooth muscle of other visceral organs, together with the clinical observation of both distention and spasm of vessel walls, suggests that true visceral pain may arise from blood vessels. This reasoning by analogy is supported by experimental and clinical evidence presented in this paper.

EXPERIMENTAL EVIDENCE OF PAIN FROM BLOOD VESSELS

Spiegel and Wassermann[7] observed that slight distention of the ascending aorta elicited pain reactions in experimental animals. Odermatt[8] demonstrated that pain reactions may be elicited by distention of arteries of any caliber. Bazett and McGlone[9] showed that simple puncture of the wall of an artery was painful. They asserted that the pain was characteristically of a dull, aching and unbearable quality, frequently accompanied by uncontrollable reflex reactions, such as sweating, sudden sensations of warmth or cold, faintness or even loss of consciousness. Various investigators have shown that when a spasm of an artery is produced by injection of barium chloride a very intense pain invariably results. Odermatt[8] was able to elicit pain reactions by various manipulations of blood vessels and attributed the pain to change in tension acting on the perivascular endings of visceral afferent neurons. He concluded that pain reactions following injection of irritating substances arose in the capillaries. He was inclined to regard the spasm of blood vessels and the resultant pain as comparable with intestinal colic.

OBSERVATIONS OF SIMILAR PHENOMENA IN SURGICAL PROCEDURES

In the injection treatment of varicose veins I have noted two types of pain that appear to arise in the vein walls rather than from somatic tissues. The first is a slight aching sensation elicited most commonly when a small group of varices is isolated by pressure from connecting veins and the fluid put in under pressure so as to distend the vein. The pain begins during the injection and is relieved promptly by withdrawing some of the fluid into the syringe so as to lessen the tension in the vein. The second type of pain is elicited when certain irritating

solutions, such as a salt-sugar mixture, are used and the injection made with the patient standing. This pain is described as a deep, aching pain. It comes on after the injection is complete, radiates down the leg along visible venous channels, and not uncommonly these channels may be observed to diminish in size coincident with the onset of pain having a definite wave-like character. The time factor in the appearance and duration of this pain is of a remarkable constancy. The pain begins gradually about 70 seconds after the injection is begun, increases in severity for about forty-five seconds, maintains its height for a little over a minute and then fades, so that at the end of four minutes from the time of injection the pain has practically disappeared. The absence of evidence of leakage around the needle together with the wave-like character of the pain seems to indicate that the pain is due to a spasm of the smooth-muscle elements in the vein walls.

During operations under novocain anesthesia the patient may complain of pain when an artery is clamped. Ligation of the main gastric arteries is almost invariably painful. The pain is often intense, usually of short duration and described as diffuse rather than being accurately localized. If an artery showing this sensitiveness is stretched laterally or pulled upon, the patient will frequently complain of pain. On several occasions I have noted pain from clamping arteries in an operative field in which the local anesthesia seemed otherwise entirely satisfactory.

Recently during an operation under spinal anesthesia in which the anesthesia was incomplete, it was noted that the patient seemed to experience but little pain when the skin was incised, but complained bitterly when an artery was clamped and ligated.

CUTANEOUS STIMULATION FOR RELIEF OF VISCERAL PAIN

As far back as medical history extends we find records of the use of various forms of cutaneous stimulation for the relief of deep-seated pain. Rubbing, heat, cold, cuppings, leeches and counter-irritants of all kinds have been utilized in the treatment of visceral pain. Perhaps the commonest form of cutaneous stimulation is the application of heat over the painful area. We know from actual experiment that the penetration of heat is not great, yet the patient frequently experiences a marked and prompt lessening of his pain. Nor is it reasonable to assume that there occur any immediate changes in the underlying inflammatory lesion. It is more likely that any effect on the pain produced by the external application of heat is due to reflex changes. Wernoe[10] not only showed that visceral stimulation may bring about viscero-cutaneous and vaso-motor reflex changes in the periphery but also demonstrated that cutaneous stimulation was capable of eliciting vaso-motor reactions in the vessels of the viscera. In some of his experimental animals cutaneous stimulation resulted in reflex visceral hyperemia and in others a visceral ischemia.

The prompt change in the character of the visceral pain following cutaneous stimulation, together with the observation that immediate vascular reflexes may occur, seems to argue that certain types of visceral pain may originate in the walls of blood vessels. This argument is by no means conclusive, since we know that smooth muscle of other organs may be reflexly affected by the cutaneous stimulation, yet the argument remains worthy of consideration.

ISCHEMIA AS A CAUSE OF PAIN

Many clinical conditions associated with pain are characterized by localized arterial spasm with a resultant ischemia of the surrounding tissues. This ischemia is commonly held to be the cause of the pain. While it is impossible in the present state of our knowledge to rule out ischemia as a contributing factor in certain cases of pain, I have never been able to convince myself that it is the major factor in many of the pain syndromes for which it has been held responsible.

If one applies an Esmarch bandage and then a tourniquet to his arm for ten or fifteen minutes there is no real pain experienced in spite of the presence of a marked ischemia and a developing cyanosis. One feels a sensation of weight and numbness to which is added, within a few minutes, a fine, vibratory, tingling sensation in the fingers, but no real pain is experienced until the tourniquet is released. Then one feels a rather painful sense of suffusion and sees the extremity flush suddenly as the capillaries fill, followed by stabbing pains that resemble sharp, scattered and intermittent electric shocks.

When an extremity is exposed to extreme cold there follows an ischemia associated with an aching pain. If the pain were entirely due to the ischemia one might expect it to disappear when the extremity was warmed. Experience indicates, on the contrary, that a tingling pain appears, coincident with the return of the circulation, that is often characterized by a painful throbbing sensation which is synchronous with the pulse rate.

PAINFUL SYNDROMES

1. *Raynaud's Disease.* Raynaud's disease is a clinical syndrome characterized by vascular changes in the hands or feet, usually brought on by exposure to cold. The involved extremities become cold, painful and increasingly cyanotic, and, in severe cases, nutritional changes may result in a progressive, dry gangrene of the terminal phalanges. The current explanation of the pain in this syndrome is that it is due to chemical changes in the tissues resulting from the ischemia, which act on the somatic sensory nerve endings to produce pain impulses.

We know little of the underlying mechanisms of this syndrome. Sir Thomas Lewis[11] maintains that the essential factor is a local disease of the digital vessels and holds that vaso-motor reflexes have little or nothing to do with the attacks. If this concept is correct, it naturally throws doubt upon the rationality of the various surgical sympathec-

tomies proposed for the relief of Raynaud's syndrome. Yet, in Lewis' own paper he presents a case which seems to me to throw doubt upon his interpretations. This woman (case 1, Observation 33) developed a typical attack following the exposure of a single finger to cold. The middle finger of the right hand was plunged into cracked ice, while the remainder of the fingers were kept at room temperature. Severe pain developed, followed by a rapidly increasing cyanosis of the fingers of *both* hands. Lewis explains this attack as resulting from the pain stimulation of that particular finger and seems to think that the other fingers were in an abnormal state at the time of the experiment, However, I am unable to see why pain should be accepted by Lewis as a more adequate stimulus for reflex precipitation of an attack in preference to any other unperceived impulse from the vessels or the surrounding tissues of the finger. In fact, it would seem to me that if reflexes could produce the attack under any circumstances, Lewis' position would be materially weakened.

I am inclined to interpret the attacks of Raynaud's syndrome as due to reflexes by way of visceral pathways which result in vascular spasm, and believe that the resultant pain originates in the walls of the vessels. Kuntz[12] agrees with this opinion and says in a personal communication, "It is my opinion that in such conditions as Raynaud's disease the pain is due to spastic contraction of the arteries rather than to lack of blood to the tissues."

2. *Angina Pectoris.* Angina pectoris is another pain syndrome in which the underlying factors are but little understood. It is commonly held that the pain is due to ischemia of the contracting cardiac musculature following spasm of the coronary arteries. The areas to which the referred pain is distributed, however, would seem to point toward the base of the aorta or the coronary arteries as the origin of the pain, rather than the myocardium. I am of the opinion that the pain arises in the walls of the vessels themselves. Certain results of surgical sympathectomy would appear to confirm this opinion.

Most of the surgical procedures, such as those advocated by Jonnesco and Leriche are proposed as palliative measures in that they aim to interrupt afferent pathways from the heart. On the other hand, Coffey and Brown[13] report favorable results from simple extirpation of the superior cervical ganglion. Since the available anatomical evidence ascribes a purely motor function to this ganglion, the only possible explanation of pain relief from this procedure would be that it had prevented motor mechanisms responsible for the pain. The known afferent pathways from the heart by way of the middle and inferior cardiac nerves being intact, there should be no reason why the subject should not experience pain if the causative mechanism still existed after the Coffey operation.

Any case, then, of pain relief in angina pectoris resulting from extirpation of the superior cervical ganglion would confirm the belief

that in that particular instance the attacks had been the result of vascular spasm, and, since the pain pathways remain intact, that the pain was the direct or indirect result of the smooth muscle spasm.

3. *Gastric Ulcer.* Odermatt[8] says, "The pain in gastric ulcer is possibly to be explained by a chemical stimulation of the arteries and veins." This suggestion is interesting but in itself does not appear to be consistent with the periodicity and certain other peculiarities of ulcer pain. Kinsella[14] ascribed the pain of peptic ulcer to pressure changes on the inflamed tissues of the ulcer bed. He showed in an ingenious manner that those conditions which give rise to ulcer pain always produce congestion and vascular changes in the ulcer area. If this be true, it would seem likely that at least a part of the pain might arise in the walls of the vessels, particularly in the presence of inflammation and when the ulcer tissue around the vessels is indurated.

4. *Emboli.* Emboli may cause pain. Certain emboli such as fat and tumor cells may not cause pain, but a large embolus, lodging for instance in a mesenteric artery can give rise to most severe pain and a high degree of "shock." Even small emboli to the brain or lungs may be accompanied by severe pain. That the pain is not due to the resultant infarction is evidenced by the fact that infarcts are frequently found at autopsy when there has been no history of pain. Nor is the pain due to inflammatory changes produced by a lodged embolus since the pain is immediate in onset. It appears more probable that the pain is due to a sudden arrest of the embolus at a vessel bifurcation with an immediate distention at the point of impact and a distending back-pressure in the vessel proximal to the obstruction. It is also possible that a reflex spasm of the vessel results, in much the same fashion that spasmodic expulsive efforts follow obstruction of the lumen of the intestine.

5. *Headaches.* Headaches of various types may be due to over-distention or spasm of blood vessels. After the use of amyl nitrate or nitroglycerin to lower blood pressure, or, as I have noted after the intravenous injection of corpus luteum, the patient exhibits a superficial vaso-dilatation accompanied by a severe, throbbing headache, The pain usually disappears with the fading of the visible vaso-dilatation within four or five minutes. The headache is probably the result of vaso-dilatation within the unyielding calvarium, and may be ascribed to the pressure on the meninges or the increased tension in the walls of the blood vessels themselves.

6. *Polyarthritis.* From the Mayo clinic[15] we are getting interesting reports of the results of ganglionectomies for polyarthritis. The cases usually present a pre-operative picture of cold extremities, marked sweating, tender and swollen joints, trophic changes in the muscles, skin and nails, combined with severe pain. Rowntree and Adson[15] comment that in this type of arthritis there is a hyperactivity of the sympathetic nervous system characterized by marked vascular disturb-

ances and sweating. Following operation these cases show surprising clinical improvement, perhaps the most striking feature being the relief of pain accompanying the improvement in circulation. The use of Brown's[16] "vaso-motor index" was based on the observation that the pain was less severe or absent, during fever when there was an improved circulation.

All of these observations, perhaps even the fact that arthritic patients experience variations in pain with changes in weather and altered barometric pressures, point to a relationship of the pain to vascular alterations. I am of the opinion that at least a part of the pain accompanying arthritis may arise in the walls of blood vessels.

DISCUSSION

Surgery directed toward a division or extirpation of portions of the visceral nervous system now commands a remarkable degree of interest. Peri-vascular sympathectomies, in spite of a lack of rational anatomical explanation, continue to yield favorable results in the hands of experienced operators; operations on the cervical sympathetics for angina pectoris; ramisections and ganglionectomies for spastic paralysis, pain of inoperable carcinoma, painful amputation of stumps, causalgia, Hirschsprung's disease, polyarthritis, Raynaud's disease and even certain cases of Buerger's disease and senile gangrene, are being reported in increasing numbers. In many of these conditions the major indication for surgery is the intractable pain, and, as Leriche[17] indicates, there is a definite field for this "Chirurgie de la douleur." As yet there is no unanimity of opinion as to the indication for operation, no standardization of technic and no agreement as to the underlying mechanisms responsible for the pain. It is my conviction that the blood vessels themselves are directly responsible for the pain in many instances.

When the time comes that we can state with assurance the rôle of the blood vessels in the causation of pain and disability for a given case, then surgery of this type will have a rational basis, whether directed toward the division of afferent neurons conducting pain impulses, or, more reasonably, toward division of autonomic fibers responsible for the functional abnormalities of the blood vessels. It would remain but to select the particular surgical procedure that most completely interrupts the visceral pathways to the affected part, with the least risk to the patient.

SUMMARY

Spasm and distention of smooth muscle in the walls of hollow viscera have been established as "adequate" stimuli for true visceral pain. The blood vessels exhibit these conditions in the same manner as the internal viscera and are provided with afferent pathways for the conduction of pain impulses. Experimental evidence is presented to

demonstrate that pain may arise from abnormal degrees of spasm or distention of the walls of blood vessels. Instances are cited illustrating surgical sensitiveness of arteries and conditions giving rise to pain in veins. A number of clinical syndromes characterized by pain are mentioned, in which afferent impulses of pain may originate in the walls of blood vessels.

REFERENCES

1. Mackenzie, Sir James: Angina Pectoris, pp. 22-28, Oxford Medical Press, 1923.
2. Hurst, A. F.: The Sensitivity of the Alimentary Canal in Health and Disease. Lancet, **1**, 1051, 1119, 1187, 1911.
3. Ryle, J. A.: Visceral Pain and Referred Pain. Lancet, 1, 895, 1926; Clinical Study of Pain. British Med. J., **1**, 537, 1928.
4. Poulton, E. P.: Experimental Study of Certain Visceral Sensations. Lancet, **2**, 1223, 1277, 1928.
5. Cannon, W. B.: Some Aspects of the Physiology of Animals Surviving Complete Exclusion of Sympathetic Nerve Impulses. Am. J. Physiol., **89**, 84, 1929.
6. Carlson, A. J., and Luckhardt, A. B.: Studies on the Visceral Sensory Nervous System. Am. J. Physiol., Vol. 54 and 55 from Nov. 1920 to Sept. 1921.
7. Spiegel, F. A., and Wassermann, S.: Experimentelle Studien über die entstehung des Aortenschmerzens und seine Leitung zum Centralnervensystem. Zeitschr. f. d. ges. exp. Med., **52**, 180, 1926.
8. Odermatt, W.: Die Schmerzenpfindlichkeit der Blutegefasse und die Gefaszreflexe. Beit. z. klin. Chir. Turingen, **127**: 1, 1922.
9. Bazett, H. C., and McGlone, B.: Note on Pain Sensations which Accompany Deep Puncture. Brain, **51**, 18, 1928.
10. Wernoe, T. B.: Aestesioscpoia Abdominalis. Ugesk. f. Laeger, **82**, 1415, 1920.
11. Lewis, Sir Thomas: Experiments Relating to the Peripheral Mechanism Involved in Spasmodic Arrest of the Circulation in the Fingers, a Variety of Raynaud's Disease. (Collaboration with Wm. J. Kerr.) Heart, **15**, 8, 1929.
12. Kuntz, A.: The Autonomic Nervous System. Lea and Febiger, 1929.
13. Coffey, W. B., and Brown, P. K., and Humber, J. D.: Angina Pectoris, the Anatomy, Physiology and Surgical Treatment, Tulane University Press, New Orleans, 1927.
14. Kinsella, V. J.: The Mechanism of Pain Production in Abdominal Visceral Disease with Special Reference to the Pains of Peptic Ulcer. Med. J. of Australia, **1**, 6, 1928.
15. Rowntree, L. G., and Adson, A. W.: Results of Bilateral Lumber Sympathectomy and Ramisectomy for Polyarthritis of the Lower Extremities. J. A. M. A., **93**, 179, 1929.
—. Bilateral Lumbar and Thoracic Sympathetic Ganglionectomy and Ramisectomy for Polyarthritis of the Lower and Upper Extremities. Trans. Assoc. Am. Phys., **44**, 221, 1929.
—. The Surgical Indications for Sympathetic Ganglionectomy and Trunk Resection in the Treatment of Chronic Arthritis. Surg., Gynec. and Obstet., 204, 1930.
—. The Effects of Sympathetic Ganglionectomy and Ramisectomy in Arthritis. Amer. J. Physiol., **90**, 1929.
16. Adson, A. W., and Brown, G. E.: Physiologic Effects of Thoracic and of Lumbar Sympathetic Ganglionectomy or Section of the Trunk. Arch. of Neur. and Psych., **38**, 323, 1929.
—. Thoracic and Lumbar Sympathetic Ganglionectomy in Peripheral Vascular Diseases. J. A. M. A., **94**, 250, 1930.
—. The Treatment of Raynaud's Disease by Resection of the Upper Thoracic and Lumbar Sympathetic Ganglia and Trunks. Surg., Gynec. and Obst., 1929, **48**, 577, 1929.
—. The Surgical Indications for Sympathetic Ganglionectomy and Trunk Resection in the Treatment of Diseases Resulting from Vasomotor Spasm of Peripheral Arteries. Bulletin of New York Academy of Medicine **6**, 17, 1930.
17. Leriche, R.: La Chirurgie de la Douleur. La Presse Médicale, **35**, 497, 1927.
—. Results de la Chirurgie de la Douleur. La Presse Médicale, **35**, 561, 1927.

THE RELATIONSHIP OF AURICULO-VENTRICULAR CONDUCTION TIME IN RHEUMATIC FEVER TO SALICYLATE THERAPY*

JOHN WYCKOFF, M.D., ARTHUR C. DEGRAFF, M.D.,
AND SOLOMON PARENT, M.D.
NEW YORK, N. Y.

IN 1924, Cohn and Swift[1] emphasized the fact that the heart is frequently involved in patients with rheumatic fever. They found, upon electrocardiographic study of a series of such patients, that many of them exhibited an increased P-R interval (a prolongation in A-V conduction time) when compared with the basic normal P-R interval for that particular patient.

Levy and Turner[2] (1927) stated that the lesions which are responsible for prolongation of the conduction time may be influenced by anti-rheumatic drugs. These authors observed three patients with P-R time of .20 seconds or more and other signs of active rheumatic fever. Following the institution of salicylate therapy, in addition to the usual anti-symptomatic effects, there was a gradual reduction in the P-R time to within normal limits; and conversely there occurred a prolongation of the conduction time following withdrawal of the drug. A second course of salicylate was again followed by a shortening of A-V conduction.

Master[3] (1927) showed that salicylates administered in large doses to normal individuals had no effect whatsoever upon the A-V conduction time.

Cohn and Swift[1] (1924) "noticed in one patient in whom the conduction time was .24 on the sixteenth day of disease when the temperature was 103.5° F., that 4 days later, when the temperature had fallen to 100° F., a marked increase in its duration took place to the extent that dropped beats (incomplete heart-block) were observed. At this time she was taking neocinchophen.† It is inferred that neochinchophen was not responsible for this occurrence because of the fact that later, although the exhibition of the drug was continued, the conduction time fell to limits which, for this patient, were considered normal."

Carr and Reddick[5] (1928) in a study of conduction disturbances in acute rheumatic infections report that of the nine patients who developed A-V block while on sodium salicylate (4-8 gm. daily) eight patients showed a gradual return to normal A-V conduction time, while

*From the Third (New York University) Medical Division of Bellevue Hospital and Department of Medicine, New York University.

†The effects of cinchophen in rheumatic fever are precisely the same as those of salicylates.—Hanzlik[4].

the same dosage was continued; and the other retained the conduction disturbance long after discontinuance of the drug. Their observations led them to believe that the salicylates are without effect on conduction disturbances, neither causing them nor tending to control them.

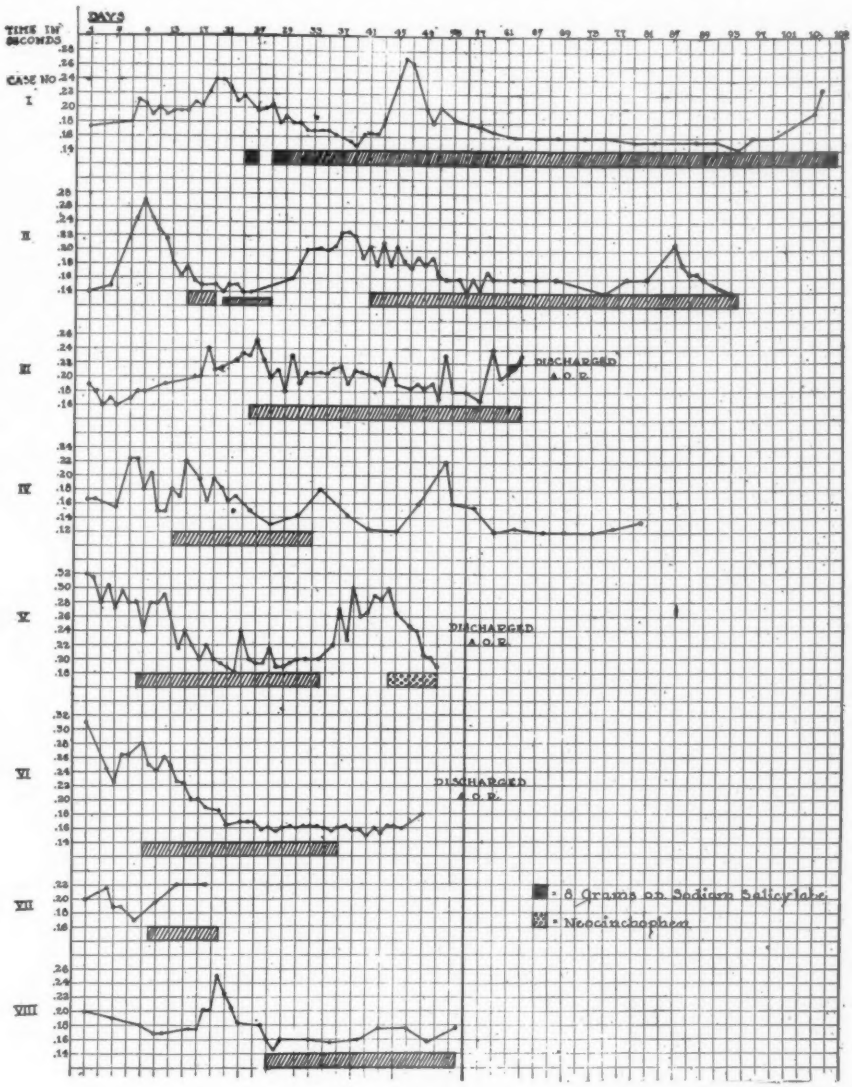


Fig. 1.—Relationship between variations in A-V conduction time and salicylate medication during the course of rheumatic fever. Time in days is plotted along the abscissa. The P-R interval in seconds is plotted along the ordinate.

In contrast to this, are the conclusions of Levy and Turner that "the administration of salicylate exerts a favorable effect upon the lesions in the heart muscle" as evidenced by the effect on A-V conduction time. Since this conclusion is of such far reaching significance and

has been challenged by Carr and Reddick, it was thought advisable to repeat this work upon a group of patients carefully controlled and observed for a long period of time.

METHOD

Every patient having rheumatic fever was electrocardiographed routinely twice a week. If any of these patients showed a P-R interval of .20 seconds or more, tracings were taken every day. Alternate

TABLE I

CASE NO.	AGE	SEX	DIAGNOSIS*
1	14	F.	A. Rhematic fever, inactive and active B. Enlarged heart, mitral stenosis and insufficiency, aortic insufficiency, acute myocarditis C. Sinus tachycardia, auricular and ventricular premature contractions, first stage A-V block D. Class II
2	18	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
3	34	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
4		F.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis and pericarditis C. Sinus tachycardia, first and second stage A-V block D. Class II
5	47	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first and second stage A-V block D. Class II
6	29	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first and second stage A-V block D. Class II
7	23	M.	A. Rheumatic fever (polyarthritis) inactive and active B. Enlarged heart, mitral stenosis, mitral insufficiency, aortic insufficiency, acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
8	32	M.	A. Rheumatic fever (polyarthritis) inactive and active B. Enlarged heart, mitral stenosis and insufficiency, aortic insufficiency, acute myocarditis C. Sinus tachycardia, acute myocarditis, auricular premature contractions, first stage A-V block D. Class II Peritonitis
9	24	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis, enlarged heart (x-ray) C. Normal sinus rhythm, first stage A-V block D. Class II

*Diagnosis conforms to the criteria adopted by American Heart Association.

cases showing increased A-V conduction time were given salicylates after a control period of one week. The dosage (8 grams per day) was just under the average toxic dose (10 grams per day) determined by Hanzlik.[4] That this was sufficient was evidenced by the fact that four patients showed toxic symptoms on this dose (cases 1, 2, 4, 5). Incidentally, all of the eight treated patients exhibited a prompt decline in temperature and alleviation of joint symptoms with the institution of salicylate therapy. The temperature stayed down while the patients

TABLE I—CONT'D

CASE NO.	AGE	SEX	DIAGNOSIS*
10	24	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
11	33	F.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis, acute pericarditis C. Normal sinus rhythm, first stage A-V block D. Class II
12	24	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
13	22	F.	A. Rheumatic fever (polyarthritis) inactive and active B. Enlarged heart, mitral stenosis and insufficiency, acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II Acute sero-fibrinous pleuritis
14	44	M.	A. Rheumatic fever (polyarthritis) inactive and active B. Enlarged heart, mitral insufficiency, aortic insufficiency, acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
15	17	M.	A. Rheumatic fever (polyarthritis) inactive and active B. Enlarged heart, mitral stenosis and insufficiency, acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
16	20	M.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
17	47	M.	A. Rheumatic fever (polyarthritis) inactive and active B. Enlarged heart, mitral stenosis and insufficiency, acute myocarditis C. Normal sinus rhythm, first stage A-V block D. Class II
18	35	F.	A. Rheumatic fever (polyarthritis) active B. Acute myocarditis C. Sinus tachycardia, first stage A-V block D. Class II

*Diagnosis conforms to the criteria adopted by American Heart Association.

were taking the medication regardless of the variations in A-V conduction time. In Table I are listed the age, sex and diagnosis of all cases used for this study.

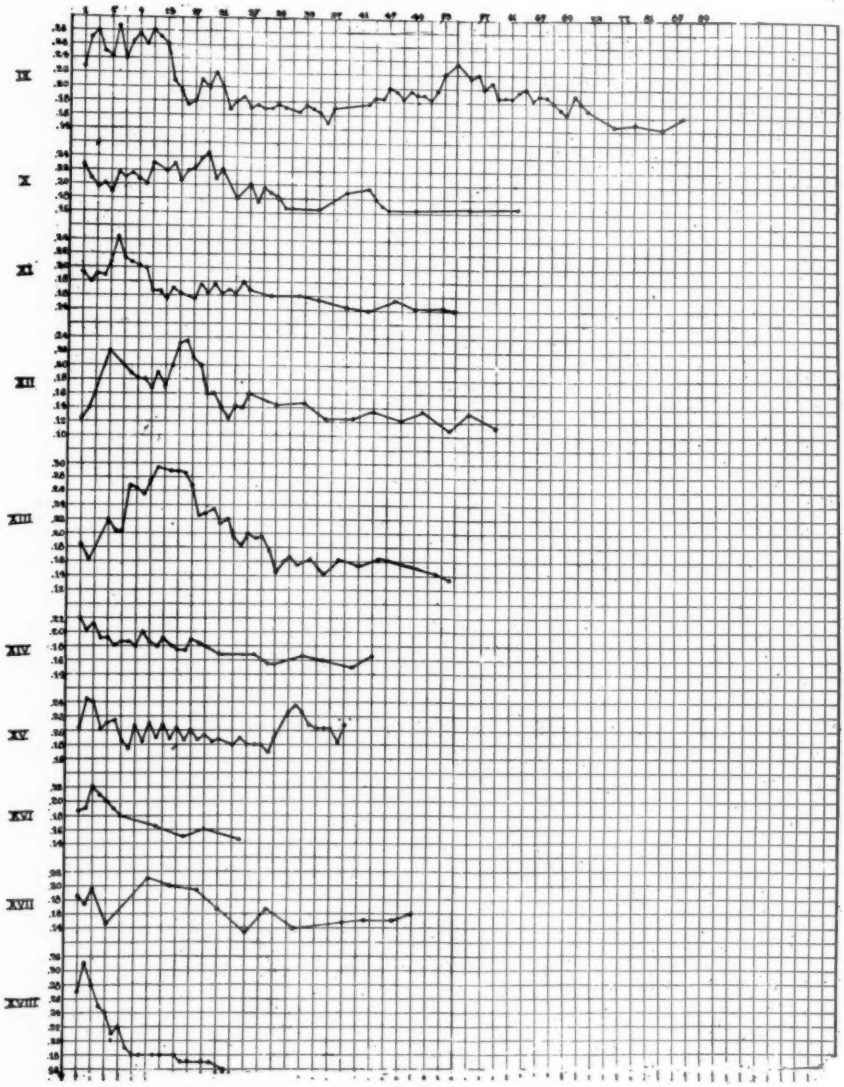


Fig. 2.—Variations in A-V conduction time during the course of rheumatic fever uncomplicated by salicylate therapy. Time in days is plotted along the abscissa. The P-R interval in seconds is plotted along the ordinate.

RESULTS

Eight patients were started on a daily dose of 8 grams of sodium salicylate following a control period of seven days after the discovery of an increased A-V conduction time. In figure 1 is plotted the rela-

tionship of variations in A-V conduction time to salicylate therapy. In two cases the P-R interval had already reached the normal for those patients before the medication was begun (cases 2 and 8). In cases 5 and 6 the P-R intervals gradually returned to normal while on salicylates. In case 5 the drug was discontinued and with this there was a subsequent increase in the P-R interval, which promptly fell to normal as the drug was resumed. This case was the only one among the eight which reacted in a manner similar to those reported by Levy and Turner. Incidentally even this case showed an increase in A-V conduction time while on the first course of salicylates. In contrast, four cases (1, 2, 3, and 7) definitely showed an increasing P-R interval while on salicylate medication. In cases 1, 2, 3, and 4 it is obvious that no relationship exists between changes in A-V conduction time and salicylate therapy.

In table 2 the variations in A-V conduction time from day to day are plotted in a control group of patients with rheumatic fever not receiving salicylate medication. A careful study of this chart shows that during the natural course of rheumatic fever uncomplicated by salicylate therapy there are wide and inconstant variations in the A-V conduction time. If antirheumatic drugs are used at any time during the disease it may easily be seen how at one time they might seem to cause an increase in A-V conduction time, at other times a decrease, and again to have no effect whatsoever. Thus if sodium salicylate had been given on the second day in case 18, when the P-R interval was .31 seconds, we would have been struck with the remarkable rapidity with which salicylates brought the A-V conduction time to within normal limits. Likewise, had salicylates been given on the second day of case 13 we should have been alarmed by the rapidity with which salicylates produced block.

CONCLUSION

Disturbances of A-V conduction time during the course of rheumatic fever show wide and inconstant variations, and there is no proof that they are influenced by salicylate therapy.

REFERENCES

(1) Cohn, A. E., and Swift, Homer: Electrocardiographic evidence of myocardial involvement in rheumatic fever. J. Exp. Med. **39**, 1, 1924.
(2) Levy, R. L., and Turner, K. B.: Variations in auriculo-ventricular conduction time in rheumatic carditis with salicylate therapy. Proc. Soc. Exp. Biol. & Med. **25**, 64, 1927.
(3) Master, A. M.: Effect of sodium salicylate on normal human electrocardiogram. Am. Heart J. **3**, 180, 1927.
(4) Hanzlik, P. J.: Action and use of the salicylates and cinchophen in medicine. Medicine. **5**, 197, 1926.
(5) Carr, J. G., and Reddick, W. G.: Conduction disturbances in acute rheumatic infections. J. A. M. A. **91**, 853, 1928.

THE VISUALIZED ESOPHAGUS IN THE DIFFERENTIATION OF LESIONS OF THE RIGHT AND LEFT HEART*

LEO G. RIGLER, M.D.
MINNEAPOLIS, MINN.

HYPERTROPHY and dilatation confined to the right auricle and ventricle are most commonly caused by congenital defects such as patency of the ductus arteriosus, pulmonary stenosis, defects in the septa, or inversion of the large vessels. Any process such as chronic pulmonary emphysema, pulmonary fibrosis, or sclerosis of the pulmonary arteries, which produces obstruction or increased pressure in the lesser circulation, may likewise be a cause of right heart enlargement and failure. Although these conditions are far less common than lesions affecting the left heart, such as peripheral hypertension or disease of the aortic or mitral valves, they occur with sufficient frequency to make their recognition of some importance.

The clinical differentiation of congenital from acquired heart lesions is often difficult. This is especially true in those adult cases in which the history of heart disease since childhood is not clearly present, a history of rheumatism in some form is obtained, and there is little or no cyanosis. Given an adult with symptoms of heart disease, a history of rheumatism, an enlarged heart over the apex of which can be heard loud murmurs, and the diagnosis of acquired disease of the mitral valve is very likely to be made. If, in such a case, the teleroentgenogram or orthodiagraphic tracing shows an enlarged heart with a marked convexity of the left median curve representing a dilated conus pulmonalis, the diagnosis of mitral disease might appear to be amply confirmed.

This clinical and roentgenographic picture may, however, be due to a right heart lesion, either congenital in origin, or secondary to some disease affecting the pulmonary circulation. Obviously there are many diagnostic factors which must be considered and frequently the correct diagnosis is apparent by the usual clinical means. At times, however, as illustrated in the cases to be reported here, the clinical diagnosis may be very uncertain.

The postero-anterior roentgenogram which is obtained in these right heart lesions is occasionally difficult to differentiate from that obtained in mitral stenosis. In the typical cases, the high position of the median left convexity and the extreme enlargement of the conus pulmonalis may indicate the congenital origin of the lesion but in many cases the distinction is not so clear.

---

*From the Department of Roentgenology of the University of Minnesota and the University Hospital, Minneapolis, Minn.

There is one distinguishing feature, roentgenologically obtained, which has not been sufficiently emphasized in the past. In mitral stenosis the left auricle is the first cardiac chamber to enlarge and usually obtains a fairly large size before enlargement of the pulmonary artery or the right ventricle occurs. On the other hand, in most congenital lesions and always in cardiac enlargements of pulmonary origin, the hypertrophy and dilatation are confined entirely to the right side of the heart; the left auricle remains normal in size. This is a striking and constant difference between these two types of cardiac lesions and may be used to differentiate them with considerable accuracy.

As has already been repeatedly demonstrated, the best method of determining enlargement of the left auricle is by roentgenographic visualization of the esophagus.[1, 2, 3, 4, 5] The latter normally lies posterior to and in close contact with the left auricle. Enlargement of this chamber will always produce a distinct compression and posterior displacement of the esophagus. The technique and application of this method have been fully described by the author in a previous publication.[3]

The following cases are reported because they illustrate very aptly the application of this method and the remarkably accurate results which were obtained in a series of unusually difficult problems in cardiac diagnosis. The clinical findings in some of these cases will be reported in greater detail later and only those findings pertinent to the subject under consideration will be reported here.

CASE REPORTS

Case 1.—Left heart lesion; mitral stenosis. (From the Pediatric Service, University Hospital.) A girl of 11 years presented the typical symptoms of cardiac failure, a history of rheumatism, and the classical physical findings of mitral stenosis and regurgitation.

The teleroentgenogram of the heart (Fig. 1) showed marked enlargement with convexity of the left median curve, the appearance being typical of mitral valvular disease. The right lateral view with the barium-filled esophagus is shown in Fig. 2. There is a marked compression and posterior displacement of the esophagus in its middle portion extending down to a point about 3 cm. above the diaphragm. This corresponds to the location of the left auricle, is due to the massive enlargement of this chamber, and is characteristic of mitral disease. Clinical and Roentgenological Diagnosis: Cardiac enlargement, massive, mitral stenosis and regurgitation type.

This case is reported for contrast with the following and represents the typical findings in a left heart lesion.

Case 2.—Right heart lesion—pulmonary sclerosis. (From the Medical Service, University Hospital—private patient of Dr. Henry Ulrich.) A woman of 26 years presented a history of cardiac symptoms coming on suddenly after childbirth. These were progressive. On physical examination dyspnea, cyanosis, dilatation of neck vessels and pulsations over the chest were observed. An enlarged heart was made out, and over it a loud systolic murmur transmitted over the whole chest and back was heard. Evidences of pulmonary congestion and peripheral edema were present.

She was examined at different times by a number of clinicians. Two prominent cardiologists made the diagnosis of mitral stenosis. She was last seen by Dr. Henry Ulrich who made a clinical diagnosis of cardiac enlargement and failure probably from a congenital defect.

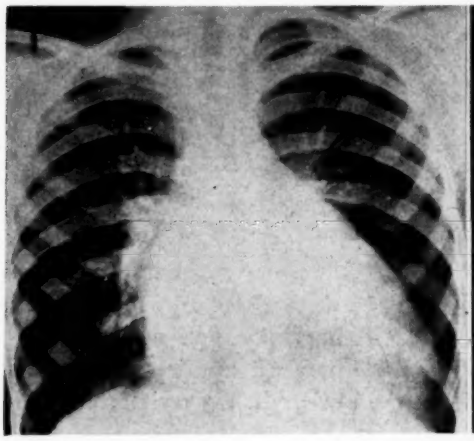


Fig. 1.

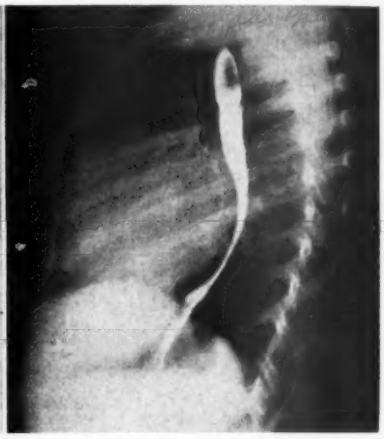


Fig. 2.

Fig. 1.—Case 1. Postero-anterior teleroentgenogram. Shows marked cardiac enlargement of the typical mitral type. Note bulging of conus pulmonalis (left median convexity).

Fig. 2.—Case 1. Right lateral view with barium-filled esophagus. Shows marked compression and posterior displacement of the esophagus in its middle third in the region of the left auricle. This indicates massive enlargement of the left auricle.

Fig. 3.

Fig. 4.

Fig. 3.—Case 2. Postero-anterior teleroentgenogram. Shows marked cardiac enlargement with extreme bulging of conus pulmonalis. Note high position and extreme convexity of left median curve. Note markedly dilated pulmonary vessels.

Fig 4.—Case 2. Right lateral view with barium-filled esophagus. Note the straight course without displacement or compression indicating no enlargement of the left auricle. Note space between spine and esophagus.

The teleroentgenogram (Fig. 3) shows a marked cardiac enlargement with extreme bulging of the left median curve representing a dilated conus pulmonalis. A notable dilatation of all the larger, and even some of the smaller, pulmonary vessels is shown, and on roentgenoscopic observation, a striking pulsation in these

vessels could be made out. The appearance is fairly characteristic of right heart enlargement but might be simulated by an extreme degree of mitral stenosis. Fig. 4 shows the right lateral view with the barium-filled esophagus. It will be noted that it runs straight through the thorax, shows no posterior displacement, and maintains its separation from the spinal shadow. This indicates the absence of left auricular enlargement, rules out conclusively mitral stenosis as a cause of such a marked cardiac enlargement, and confirms the impression of right heart enlargement, probably congenital.

Post-mortem examination was made by Dr. B. J. Clawson. The heart weighed 425 grams and showed marked hypertrophy confined to the right ventricle which was also much dilated. The other chambers and all the valves were normal; there were no congenital defects. The pulmonary arteries showed extreme arteriosclerosis with extensive dilatation. The lumina of many of the small arteries were almost closed. The other findings were of no importance.

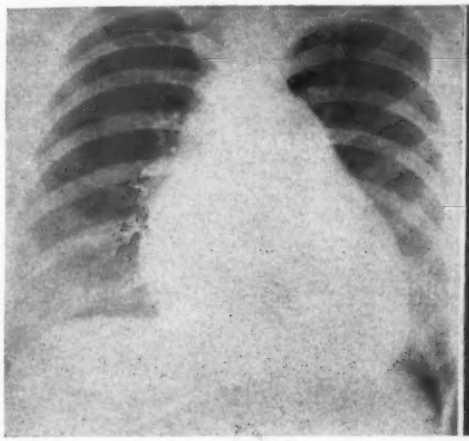


Fig. 5.

Fig. 6.

Fig. 5.—Case 3. Postero-anterior teleroentgenogram. Shows marked cardiac enlargement with extreme bulging of conus pulmonalis. Note high position and extreme convexity of left median curve.

Fig. 6.—Case 3. Right lateral view with barium-filled esophagus. Note the straight course without displacement or compression indicating no enlargement of the left auricle. Note space between spine and esophagus.

Various Clinical Diagnoses: (1) Congenital defect with cardiac enlargement. (2) Mitral stenosis with cardiac enlargement.

Roentgen Diagnosis: Right heart enlargement, probably congenital defect.

Post-mortem Diagnosis: Hypertrophy and dilatation right ventricle. Pulmonary sclerosis.

Case 3.—Right heart lesion—pulmonary hypertension? (From the Medical Service, University Hospital.) A woman of 24 years presented the clinical picture of cardiac failure coming on suddenly after excessive exertion. There was a doubtful history of rheumatism. Repeated peripheral edema was present but there were few signs of pulmonary congestion. Physical findings were variable. The heart was enlarged but murmurs were entirely absent at times, or only faintly heard and were not at all typical. Various clinical diagnoses were made including a definite diagnosis of mitral stenosis by two cardiologists.

The teleroentgenogram (Fig. 5) shows considerable cardiac enlargement with bulging of the left median curve. The high position and marked enlargement

of the conus suggests a congenital heart but the picture bears a close resemblance to that of marked mitral stenosis. Fig. 6 is the right lateral view with barium-filled esophagus which is quite normal. There is no left auricular enlargement, which ruled out mitral stenosis as a cause of the cardiac enlargement.

Post-mortem examination was made by Dr. W. A. O'Brien. There was a marked enlargement of the heart entirely confined to the right ventricle and right auricle. The remaining chambers were normal and the valves were normal; there were no congenital defects. The pulmonary arteries were dilated and showed some patches of sclerosis in the large branches. The smaller branches were, however, normal. The other findings were of no significance.

Various Clinical Diagnoses: (1) Mitral stenosis and regurgitation with cardiac enlargement. (2) Congenital defect and mitral stenosis with cardiac enlargement. (3) Congenital defect with cardiac enlargement. (4) Pulmonary sclerosis with cardiac enlargement.

Roentgen Diagnosis: Right heart enlargement, probably congenital defect.

Post-mortem Diagnosis: Right heart hypertrophy and dilatation. Possible pulmonary hypertension(?).

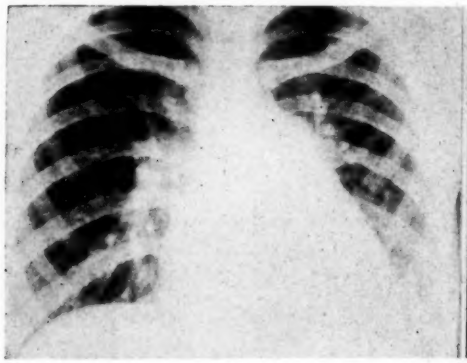


Fig. 7.

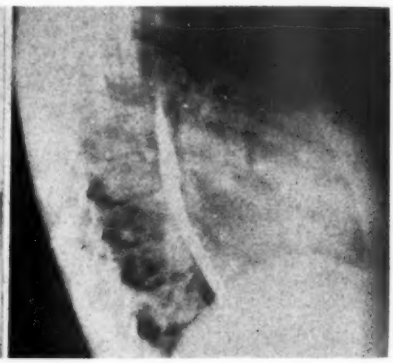


Fig. 8.

Fig 7.—Case 4. Postero-anterior teleroentgenogram. Shows marked cardiac enlargement similar to Fig. 1 and fairly characteristic of mitral disease of high degree. Note marked bulging of pulmonary conus.

Fig. 8.—Case 4. Right lateral view of esophagus. Shows slight displacement in middle third indicating very moderate left auricular enlargement. (Compare with Fig. 2.)

Case 4.—Right and left heart enlargement—mitral stenosis and absent interauricular septum. (From the Medical Service, University Hospital.)

A woman of 27 years presented the clinical picture of cardiac failure. Symptoms first began early in life following an attack of rheumatism and recurred frequently. The findings indicated both pulmonary congestion and peripheral edema. Physical findings showed a large heart of the mitral type, with a loud systolic murmur over the apex. Three clinicians disagreed as to the diagnosis as the physical findings were variable and difficult to interpret.

The teleroentgenogram (Fig. 7) shows a considerable degree of cardiac enlargement with marked bulging of the conus pulmonalis. The appearance is typical of either an extreme degree of mitral stenosis or a right heart enlargement of congenital origin. The right lateral view of the barium-filled esophagus (Fig. 8) shows only a small posterior displacement in the region of the left auricle (compare with Figs. 2 and 6) indicating only a very moderate enlargement of the latter. Considering the enlargement of the heart and the marked dilatation of the conus pulmonalis, the left auricle seemed disproportionately small if the whole process

were due to mitral stenosis. Accordingly (without any knowledge of the clinical findings) a roentgenologic diagnosis of mitral stenosis and a right heart lesion, probably a congenital defect, was made.

Post-mortem examination was made by Dr. W. A. O'Brien. The heart was markedly enlarged almost entirely on the right side. The left ventricle was, if anything, smaller than normal. The left auricle was moderately dilated and hypertrophied. The right ventricle and right auricle were massively enlarged. A high grade mitral stenosis probably of rheumatic origin was found. The interauricular septum was almost entirely absent. As a result the stenosis of the mitral valve affected the right auricle more than the left, the deficient septum preventing much increase in pressure in the left auricle. The right ventricle, however, responded to the double handicap of mitral stenosis and patent interauricular septum. The remaining findings were of little importance.

Various Clinical Diagnoses: (1) Mitral stenosis with cardiac enlargement. (2) Congenital defect with right heart enlargement. (3) Mitral stenosis with cardiac enlargement and possible congenital defect.

Roentgen Diagnosis: Mitral stenosis and congenital defect with enlargement of right heart and left auricle.

Post-mortem Diagnosis: Massive cardiac enlargement chiefly in right heart with moderate dilatation of left auricle. Mitral stenosis, probably rheumatic. Absent interauricular septum.

COMMENT

These four cases illustrate very well the possibilities of improved roentgen diagnosis of cardiac lesions with the aid of the barium-filled esophagus. A comparison of the postero-anterior films in all four cases shows a distinct similarity, while the films of the esophagus show marked differences under the varying conditions. The total heart size in Case 1, for example, is about the same as that in Cases 3 and 4. Yet in Case 1 there was marked esophageal displacement, in Case 3 no displacement and in Case 4 only slight displacement. These findings correspond perfectly with the respective sizes of the left auricle in these cases. In mitral disease (Case 1) with a fairly large heart we get extreme esophageal displacement and compression; in right heart lesions (Cases 2 and 3) no displacement; in combined mitral disease and congenital defects (Case 4) we get esophageal displacement which is far too small in proportion to the amount of cardiac enlargement.

The last three cases presented difficult diagnostic problems as indicated by the variety of clinical diagnoses. The close correspondence of the roentgen findings to the gross pathological findings is notable and indicates the accuracy of this method.

Thanks are due Dr. Henry Ulrich for the privilege of reporting the findings in Case 2.

SUMMARY

Four cases of cardiac disease are reported, in three of which post-mortem examinations were done and in the other of which the findings were unquestionable. These cases serve to illustrate the importance of the visualized esophagus in the differentiation of lesions of the right and left heart.

In a case of mitral disease the esophagus was displaced posteriorly and compressed by the enlarged left auricle.

In two cases of right heart enlargement from increased pressure in the pulmonary circulation the esophagus was not displaced indicating no enlargement of the left auricle.

In a case of mitral stenosis superimposed upon a congenital defect—absence of the interauricular septum—the esophagus was displaced slightly but much less than would be the case if the cardiac enlargement would have been due entirely to mitral disease.

The postero-anterior roentgenograms of the heart in mitral stenosis may resemble those of congenital and other right heart lesions closely. The visualized esophagus, in determining the presence or absence of left auricular enlargement, may aid greatly in making a correct diagnosis.

REFERENCES

1. Kovacs and Stoerk: Wien. Klin. Wchnschr. **23**: 1471, 1910.
2. Gäbert, E.: Fort. a. d. Geb. d. Röntg. **23**: 410, 1924.
3. Rigler, L. G.: Amer. Jour. of Roent. and Radium Therapy **21**: 563, 1929.
4. Steel, D.: Ann. Int. Med. **1**: 302, 1927.
   Amer. Jour. of Roent. and Radium Therapy **21**: 220, 1929.
5. Steele and Paterson: Am. Heart J. **4**: 692, 1929.

THE "LATENCY THEORY" OF HEART-BLOCK AND INTERPOLATED VENTRICULAR PREMATURE BEATS*

RICHARD ASHMAN, PH.D.
NEW ORLEANS, LA.

FOR more than twenty years there have been two opposing views which seek to explain the typical phenomena of partial heart-block. One, which may be termed the "latency theory," was proposed by Erlanger,[1] modified and developed by Straub[2] and Straub and Kleeman[3] and is at present held, in a different form, by Mobitz.[4, 5] According to this conception, the sinus impulse travels with normal velocity throughout the conducting tissues, but, upon reaching the junction between specialized tissue and ventricular muscle, the impulse produces a response of the ventricles only after a shorter or longer delay. The length of the delay is dependent upon the extent to which the ventricular muscle has recovered from the refractory state produced by its previous response and the strength of the impulse arriving by way of the junctional tissues. Damage in the A-V node or bundle was in general believed not to slow but to weaken the impulse. The theory, stated in this form, had to be abandoned when it was learned that an impulse, passing an injured region, will at once regain its normal strength in the undamaged tissue distal to the injury.

Mobitz[5] calls attention to the fact that the A-V node of the calf's heart consists of two portions, an auricular and a ventricular. Recognizing the difficulty in the theory as developed by Straub, he insists that the latency of the ventricular portion of the node determines the delay, and not the latency of the ventricular muscle. The sinus impulse arrives at this junction between the parts of the A-V node and, depending upon the excitability of the ventricular portion, the delay is longer or shorter.

The phenomena of partial heart-block with dropped beats as pictured by Mobitz[5] are illustrated in Figure 1. Sinus impulse *1* travels with normal velocity to the junction. After a slight latency the ventricular portion of the node lying just distally responds. The impulse then proceeds, with normal velocity, to the ventricles. The latency is short in this case because the previous beat was dropped and there has been a long recovery period. The width of the area in black may be taken as representing the duration of the absolutely refractory period of the

*From the Laboratory of Physiology, Tulane University, School of Medicine, New Orleans.

tissue distal to the junction. Impulse *2* now arrives at a much earlier stage in the recovery of the ventricular part of the node. The P-R is, therefore, considerably longer. As will be readily understood from the figure, the consequence is that impulse *3* arrives at a still earlier phase of recovery and the latency is still greater. Impulse *4* arrives to find the ventricular portion of the node unresponsive and thus a beat is dropped. With impulse *5* the sequence of events begins again.

The opposing conception (Engelmann,[6] Lewis,[7] et al.) attributes delay to slow transmission through a depressed or injured region somewhere in the auriculo-ventricular pathway. It involves either the conception of a sluggishness in the response of successive muscular elements to the oncoming impulse or of a slow rise of action current strength to an effective intensity or both. But the conception does not admit of a single greatly exaggerated delay at one point. Too great a slowing would probably spell extinction of the impulse and the ventricular response would not occur. This conception of partial block has previously been described in some detail (Herrmann and Ashman[8]).

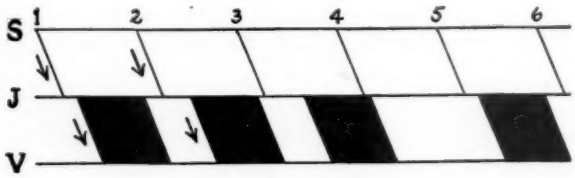


Fig. 1.—See text.

We now believe that certain details were perhaps insufficiently emphasized, while in other respects the presentation might have been simplified.

It should be pointed out that there is no intention of denying that latency at a single boundary may play a part in partial heart-block. Proof that it does so is lacking. On the other hand, there is good evidence that slow conduction is the important factor in the prolongation of the conduction time, if, in fact, it is not the sole factor.

It is not the purpose of this paper to attempt any further interpretation of partial block, but rather to present certain facts which demonstrate that the "latency theory" is not competent to explain all the observed phenomena.

Lewis[7] calls attention to a fact which he regards as in itself sufficient to rule out Mobitz' theory, namely, that conduction through a homogeneous strip of cardiac muscle is slowed by depression of any sort such as compression or increase in hydrogen-ion concentration. There is, therefore, no good reason to deny that depression caused by disease in the human heart cannot slow the impulse and lengthen the

P-R. Mobitz[5] admits that depression may slow intramuscular conduction, but argues that in partial block of the type represented there is no damage of the conducting pathway; that the effects are of vagus origin. Granting that this contention is correct, at least in many cases (e.g., over-digitalization), we must either advance other evidence against the "latency theory" or admit that it may be the correct one.

One of the first, and by no means the least formidable, of the difficulties encountered by the "latency theory" is the repeated observation of conduction times ranging from 0.5 sec. to as high, even, as 1.0 sec. (Thayer,[9] Herrmann and Ashman[8]). To explain why this is a difficulty, it is to be remembered that the action current associated with the response of each segment of muscle is generally regarded as the stimulus arousing the next successive segment to activity. When the impulse reaches the auricular portion of the A-V node and that portion responds, its action current does not continue to flow for more than about 0.3 sec. Variations in this duration depend upon the heart rate and other factors. In cases of greatly prolonged P-R intervals, therefore, according to the "latency theory," the ventricular portion of the node must respond to the stimulus from the more proximal portion anywhere from 0.1 to 0.6 sec. after the disappearance of the stimulating current.

Mobitz recognized this difficulty and was constrained to invoke some esoteric function of the nerve cells in the region of the A-V node. He is unable, however, to suggest how the nervous elements keep the stimulus going. Instead he falls back on the assertion that the typical phenomena of partial block are not obtainable in a strip of cardiac muscle where, of course, such special groups of nerve cells are not found. The answer to this assertion is that the typical phenomena of partial block *are* obtainable from strips of heart muscle.[15] Figure 2 is one of a number of examples obtained from transverse ventricular strips from the turtle heart, partial block between the ends having been induced by incisions near the center of the strip. The figure and legend are a sufficient explanation. This experiment does not, however, afford a complete refutation of the "latency theory" inasmuch as the action current at the one end of the strip had not died out at the time of response of the other end, and thus it is possible to argue that the delay represents a pause in Mobitz' sense.

Another phenomenon exhibited by the turtle heart amounts to practically crucial evidence against the "latency theory." Although there can be little doubt from the experiments of Lewis and Master[10] with the dog heart that the same behavior would be exhibited under similar conditions, the turtle heart is more suitable for experiment because its rate can be more easily controlled. Figure 3 is one example from among many. According to the usual convention, auricular beats are repre-

sented above, ventricular responses below. Abscissae represent time. Cycle lengths and P-R intervals are shown. The first series of beats (A), two in number, illustrate the well-known fact that when the recovery period is long the P-R interval is relatively short. P_1-R_1, pre-

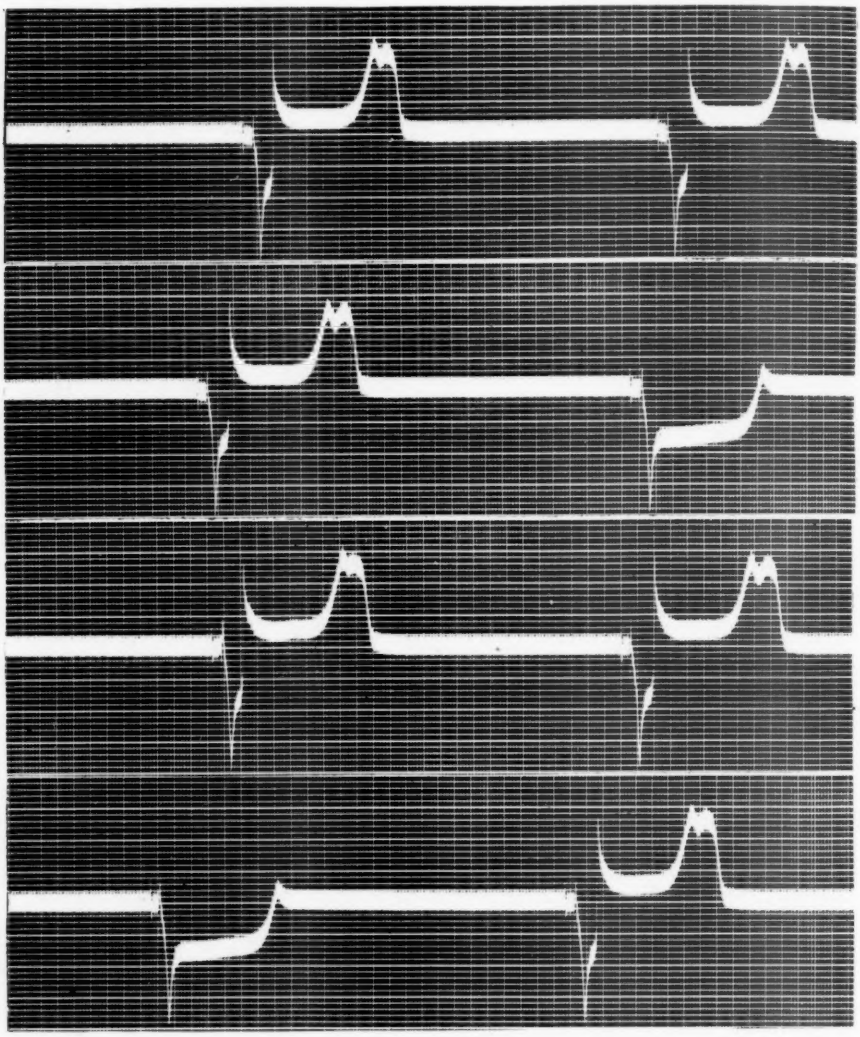


Fig. 2.—Continuous electrogram from transverse strip cut from turtle ventricle. Incisions had been made at the center of the strip leaving the two ends connected by a narrow bridge of tissue. Responses in every case are to break shocks applied to the right end of the strip. The response of this end is marked by the quick downward deflection; the response of the left end by the quick upward deflection.

The two responses, upper row, and the first response, second row, show progressive prolongation of the interval between the responses of the two ends leading up to block with the second response, second row. Following the blocked impulse the first conduction time is short (third row); the next interval is longer; the next (lower row) is blocked. Following this 2:1 block developed, but is not shown in the figure.

The time intervals in seconds between the successive responses of the right end of the strip and the ensuing conduction times (the latter in parentheses) are:

About 5.00 (0.20); 5.08 (0.224); 4.84 (0.24); 5.16 (blocked); 5.15 (0.217); 4.94 (0.24); 4.51 (blocked); 5.04 (0.23).

ceded by a rest of 10 sec., is 0.526 sec. P_3, after a 3.026 sec. rest, comes just before complete recovery in the conducting tissues and therefore P_3-R_3 is slightly prolonged (0.538 sec.).

What would have happened had another auricular impulse been interposed between P_1 and P_3? The consistent result of such interpolation is shown in the second group of responses (B). The blocked P has caused P_3-R_3 to increase from the expected 0.540 in this case to 0.652 sec., an increase of 21 per cent.

Thus an impulse, which according to Mobitz' view could not have caused a response of the structure in the turtle heart corresponding physiologically to the ventricular portion of the A-V node, did, nevertheless, greatly increase the conduction time (or "latency") for the following impulse. Now, it is well known that an ineffective stimulus does not prolong the refractory period of cardiac muscle. Consequently, it

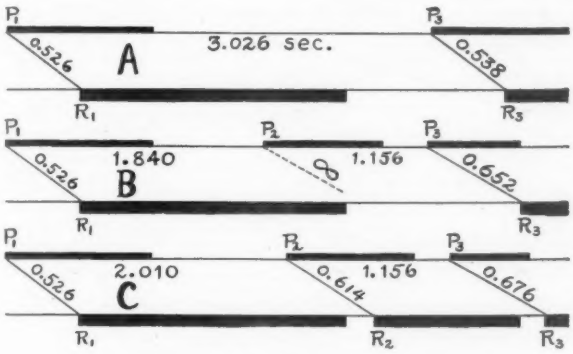


Fig. 3.—See text.

cannot be assumed that the blocked impulse affected the structures just distal to the plane of block. It was not a decrease in the excitability of these structures that was responsible for the prolonged P_3-R_3.

It may very justly be argued, however, that P_3 was weakened by the interpolation of P_2 and that this is the cause of the long P_3-R_3. In a sense, no doubt, that was true—P_3 was weakened. The long P_3-R_3 may, therefore, be due to the weakening and diminished effectiveness as a stimulus of P_3 and not to any change in the excitability of structures distal to the blocking plane of P_2.

The crucial test is to be seen in the lower group of responses (Fig. 3, C). Here P_2 is thrown in 2.01 sec. after P_1, just late enough to be transmitted. P_2-R_2 is quite long because of the short time allowed for recovery. Since the structures distal to the plane of blocking have responded, their excitability will be much lower when P_3 arrives than in the previous case where P_2 was blocked, and where P_2 cannot, therefore, be regarded as having invaded them. If Mobitz' conception is

the correct one, a P_3, thrown in at the same interval after P_2 as the P_2-P_3 interval in the second case, must either be blocked or else transmitted only after a much longer P_3-R_3 interval.

The actual result is seen in the figure. Following P_2, a P_3 is elicited at the time suggested. Therefore, the P_3 impulse will be weakened fully as much as in the former case.* Nevertheless, it will be observed that P_3-R_3 is now 0.676 sec., or only 3.7 per cent longer than it was when P_2 was blocked. Yet the structures distal to the plane of block, those corresponding functionally to the ventricular portion of the A-V node in the mammal, have had an estimated recovery period of but 0.18 sec. as contrasted with 1.23 sec. in the former instance.

The "latency theory" which attributes all the delay (barring the short conduction time proper) to latency in response of the structures distal to the plane of block, is manifestly unable to account for this result. For Mobitz to explain this result it would be necessary for him to make the highly improbable assumption that in partial block with constant auricular rate, latency plays the chief rôle, but that here, where block results from a change in auricular rate, *ventricular* latency has nothing to do with conduction time.

On the basis of the opposed conception, namely, that delay is due to slow conduction from element to element, the near equality of effect of the transmitted and blocked P_2 impulses is readily accounted for. It is only necessary to make the assumption that the blocked impulse traverses almost the entire length of the junctional tissues in which the slower conduction occurs. Thus the P_3 impulse following a blocked P_2 impulse will traverse nearly as great a stretch of relatively refractory muscle as it does when it follows a transmitted P_2 impulse. Its conduction time will, therefore, necessarily be nearly as great in the one instance as in the other.

Although, as already stated, Lewis and Master's[10] results indicate that similar relationships apply in all probability to conduction in the dog heart, yet this particular experiment was not done by them. Their results do show, however, that a blocked auricular impulse prolongs the conduction time for a following impulse, provided, of course, that the following impulse is not too late nor the blocked impulse too early. We thus possess for the dog a part, but not all, of the evidence against Mobitz' view which is so easy to obtain with the turtle heart.

Interpolated Ventricular Extrasystoles.—We have already referred to the fact that the phenomena of partial heart-block appear to be explicable without assuming the existence of an exaggerated pause at the

*Actually the weakening may be regarded as slightly greater, since the refractory phase of the muscle traversed by P_2 will in this instance be of slightly greater duration than in the former case. This follows from the fact that the P_1-P_2 interval is slightly longer than before. This fact counts all the more strongly against the "latency theory." In spite of the *greater* weakening of P_3 it goes through to the ventricle with an only slightly greater delay.

A-V junction. In interpolated ventricular premature beats, however, we generally meet with distinct, and often marked, prolongation of the P-R interval which follows the premature beat. This prolongation has been regarded by the protagonists of the "latency theory" as supporting their views.[2] For that reason we may here examine human electrocardiograms illustrating interpolated beats.

As is generally recognized, in order to explain interpolated ventricular beats at all it is necessary to suppose that the retrograde impulse from the ventricle is blocked at some point in its path back toward the auricle. Otherwise, it would cause an auricular response, or, if retrograde transmission were sufficiently slow, it would meet the oncoming auricular impulse in head-on collision. Were this latter event to occur, as it commonly does in premature beats, there would be no interpolation, since it is well known that impulses meeting on the same pathway cannot pass each other. There is mutual extinction.

Where does blocking of the retrograde impulse occur in interpolated premature beats? Briefly, it can hardly be at the junctions between ventricular muscle proper and Purkinje fibers because: (1) there is a transition in fiber structure at these junctions which presumably would make them unlikely as blocking points; (2) it is probable that the refractory period of the ventricular muscle is longer than that of the Purkinje fibers[7]; (3) one would expect the beat following the interpolated beat to be of definitely abnormal form. The last argument is particularly valid, for the prolonged P-R interval following the interpolated beat must be due to delay at and peripherally to the place where the previous retrograde impulse was blocked. If, as is usually the case, the interpolated beat arose in one or other ventricle, then, upon the arrival of the supraventricular impulse, one ventricle or region will be in a considerably more advanced stage of recovery than the other. This is a necessary consequence of the slow intramuscular conduction of the ectopic impulse. Therefore, the delay in response should be strikingly greater in one ventricle than in the other and aberrant complexes would necessarily result. Such aberration after interpolation is rarely observed. It is likewise improbable that the bundle branches are the region of blocking. Arguments (2) and (3) apply here also.

It follows, therefore, that blocking occurs either in the main stem of the bundle or within the A-V node itself. For convenience it is here assumed that the plane of block is somewhat below the middle of the node.

Figure 4 represents diagrammatically an interpolated ventricular premature beat and the supraventricular beat following it. In *A* the impulse is starting from a focus at *X* and beginning to spread through the Purkinje fibers. (See Legend.) The A-V node is represented as not yet recovered from the absolute refractory state associated with

its previous response.* In *B* the impulse spreads throughout the left ventricle, and thus the invasion of the ventricles continues until in *E*, after 0.12 sec., both ventricles have been invaded and the retrograde impulse becomes blocked low in the A-V node.† In *F*, 0.10 sec. later, an impulse starts from the sinus node. In *G* the auricles are being invaded, while the left bundle branch has recovered from its absolutely refractory state and is now relatively refractory, strongly so near the

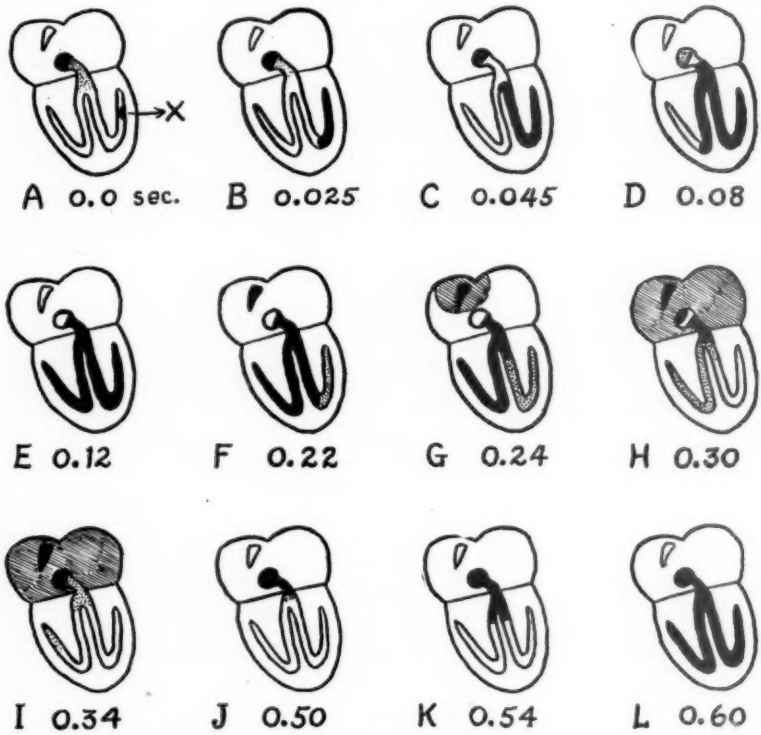


Fig. 4.—Successive stages in the invasion of the ventricles by the interpolated ventricular premature beat and by the following sinus impulse. In each diagram, the auricles are shown above, the ventricles below. The S-A (above) and A-V nodes are represented in the auricles; the A-V bundle extends from A-V node down into the ventricles where it bifurcates. No attempt is made to represent changes in the ventricular muscle proper.

Black signifies absolute refractoriness; stippled areas are relatively refractory; white areas, non-refractory.

Time from beginning of ectopic beat is shown below each heart. The velocity of spread of the impulse and other time relations are largely inferred; they are not in all cases directly determinable from the electrocardiogram. For description, see text.

---

*The moot question as to whether premature beats arise from a regularly or occasionally automatic focus or result from a circus contraction does not at all influence the argument.

†It is possible to estimate the time ordinarily required for a free-wall ventricular ectopic impulse to travel from its point of origin back to the bifurcation of the bundle. This was done by examining a number of electrocardiograms exhibiting interference between ordinary premature and supraventricular impulses. Such "combination" complexes (Wilson and Herrmann[11]) cannot occur if the retrograde impulse reaches the bifurcation before the supraventricular impulse. While recognizing that retrograde time to the bifurcation will vary with the position of the ectopic focus, with the degree of prematurity, and with the condition of the muscle, yet the average time may be taken as not more than 0.05 sec. Thus the time for retrograde transmission to the A-V node may reasonably be regarded as not more than 0.11 or 0.12 sec. Retrograde conduction to the auricles in other cases affords additional information.

bifurcation. *H* shows the further retreat of refractoriness in the ventricles, completion of invasion of the auricles, and represents invasion of the A-V node as well advanced. In *I* the retreat of absolute refractoriness is complete, and the sinus impulse has just reached the plane of retrograde block. The main bundle is still strongly relatively refractory and for that reason conduction through it is extremely slow (Drury and Regnier[12]). *J*, *K*, and *L* show the further normal invasion of the ventricles by the supraventricular impulse.*

It should be noted that from *E*, when the retrograde impulse was blocked, to *I*, when the sinus impulse arrives at the plane of block, an interval of 0.22 sec. is inferred to have elapsed. Although short, this is probably not too short a time for recovery from the absolutely refractory state in case of a response terminating as short a diastolic rest period as that preceding the premature beat. Experimental evidence for this statement will appear in a forthcoming paper by the author.

We must not at once conclude, however, that by showing conduction without a pause to be possible we have ruled out the pause. In fact, a possible difficulty presents itself. The supraventricular impulse (*I* to *K*) is regarded as advancing into tissue, each successive invaded segment of which has had a longer time for recovery from its strongly relatively refractory state. This is true, not only because the direction of conduction is now opposite to that of previous invasion by the retrograde impulse, but also because delay in conduction in the tissue traversed gives additional time for recovery of the tissue lying in advance of the active segments.

The question, therefore, arises as to whether we should expect as great a prolongation of the P-R interval following the interpolated beat as is actually observed to occur. In answer to this question, it may be pointed out that the example chosen for illustration in Figure 4 presents nearly as great a prolongation as any case found in the series examined. In one case the prolongation is considerably greater, but in this there was a disturbance in conduction which rendered it unsuitable for illustrative purposes. The P-R increased from 0.21 sec. to a maximum of about 0.43 sec. In the example illustrated in the figures, the increase is from a normal P-R of 0.19 to one of 0.32 sec. Other cases show increases of from 0.17 to 0.215 sec.; from 0.13 to 0.19; from 0.17 to 0.325; from 0.13 to 0.18; and from 0.14 to 0.19. It is clear throughout the series of electrocardiograms that the earlier the P-wave falls

*The reader may have detected an apparent inconsistency in our argument. Why is it, if, as illustrated in Figure 4, one ventricle recovers later than the other, aberration of the ventricular complex following the interpolated beat does not occur? The answer is to be found in the delay in the node and main bundle which gives all the more quickly recovering tissues below ample time for recovery? As a matter of fact some of the beats in question do show a barely perceptible deviation from the normal, a change hardly great enough to be called aberration, and not nearly so great as would be anticipated if, as discussed above, retrograde block and the observed P-R increase depended upon retrograde block of the ectopic beat in the ventricles.

with reference to the T-wave of the interpolated beat, the longer is the P-R interval. If too early, blocking occurs and the premature beat is not interpolated. It is usual to find both interpolated and ordinary non-interpolated beats in these cases.

It cannot be too strongly stressed that if the interpolated beat be of junctional origin, i.e., from a focus near the retrograde blocking point, the prolongations may be greater, and it must also be kept in mind that, because of the likelihood of aberration of very early junctional prematures, it may be impossible to distinguish them from ventricular premature beats. In such a case the ectopic impulse is not retrograde, and while the supraventricular impulse is invading the bundle there will not have been as long a time for recovery in the successively invaded segments. In such a case, too, a P-wave arising as early as in the interval between R and T may be transmitted under favorable circumstances. On the other hand, in the cases examined, where the premature beat is of strictly ventricular origin, such early impulses have invariably been blocked.

One further point may be touched upon, i.e., why is the retrograde impulse blocked? On this point we can say nothing. Equally premature impulses in some hearts may pass back to the auricles. One thing, however, seems certain and has been long recognized. Retrograde block in interpolated beats is not contingent upon damage of the conducting pathways. Nor can it be assumed that separate pathways are involved in forward and retrograde conduction (Skramlik[13]), one of which is injured. In this connection a recent paper by Ashman and Hafkesbring[14] is pertinent.

SUMMARY

Experimental evidence has been presented which shows that the fundamental postulates of the "latency theory" of partial heart-block are not valid for conduction in the turtle heart.

From an examination of interpolated ventricular premature beats it is concluded that the observed phenomena do not compel us to accept that theory for the human heart.

The author is greatly indebted to Dr. G. R. Herrmann for permission to examine and make use of the more than four thousand electrocardiograms in the Heart Station at Charity Hospital and for helpful comment upon the text of this communication.

REFERENCES

1. Erlanger, J.: Journ. Physiol., **16**: 160, 1906.
2. Straub, H.: Münch. Med. Wchnschr., **65**: 643, 1918.
3. Straub, H. and Kleemann, M.: Deutsch. Arch. f. klin. Med., **123**: 296, 1917.
4. Mobitz, W.: Zeitschr. f. d. ges. exper. Med., **41**: 180, 1924.
5. Mobitz, W.: Zeitschr. f. klin. Med., **107**: 449, 1928.

6. Engelmann, T. W.: Arch. f. d. ges. Physiol., **62**: 543, 1896.
7. Lewis, T.: The Mechanism and Graphic Registration of the Heart Beat, 3rd. Ed., London, 1925.
8. Herrmann, G. R. and Ashman, R.: Am. Heart Journ., **1**: 269, 1926.
9. Thayer, W. S.: Arch. Int. Med., **17**: 13, 1916.
10. Lewis, T. and Master, A. M.: Heart, **11**: 209, 1925.
11. Wilson, F. N. and Herrmann, G. R.: Arch. Int. Med., **31**: 923, 1923.
12. Drury, A. N. and Regnier, M.: Heart, **14**: 263, 1928.
13. v. Skramlik, E.: Arch. f. d. ges. Physiol., **184**: 1, 1920.
14. Ashman, R. and Hafkesbring, R.: Am. Journ. Physiol., **91**: 65, 1929.
15. Samojloff, A.: Arch. f. d. ges. Physiol., **222**: 516, 1929.

DIPHTHERIA AS A CAUSE OF LATE HEART-BLOCK*

STUYVESANT BUTLER, M.D., AND SAMUEL A. LEVINE, M.D.
BOSTON, MASS.

THE occurrence of unexpected cases of heart-block in otherwise healthy persons and especially the occasional instance in a child or young adult suggests that there must be some other causative factors besides arteriosclerosis, syphilis and rheumatic fever. Years ago syphilis in the form of gumma of the septal tissue was thought to be a frequent cause, and rarely gummas have been recorded, but not in nearly sufficient numbers to account for even the majority of cases of heart-block. It is undoubtedly true that arteriosclerosis is a contributing and perhaps the most important single causative factor in the majority of cases, but a number of patients, recently seen by us, in whom arteriosclerosis was conspicuous by its absence and in whom there was no evidence of syphilis, coronary artery disease, or rheumatic infection, and where there was no other obvious influence such as digitalis or fever, have led us to look farther for etiological factors. Some years ago mention was made by one of us[1] in a footnote that in several cases of Adams-Stokes disease there had been a history of early diphtheria. For this reason a study has been made to determine the incidence of a history of early diphtheria in patients with heart-block or Adams-Stokes disease.

This concept of causal relationship gains theoretical support from the pathological findings in diphtheria. Marvin and Buckley[2] described two cases of diphtheria which developed complete heart-block, as proved by electrocardiograms, during the course of the disease. In one case microscopic sections of the conduction apparatus showed edema and infiltration about the bundle of His, including both branches, as well as about the sino-auricular and auriculo-ventricular nodes. McCulloch[3] reported three proved cases of complete heart-block during diphtheria. Fleming and Kennedy,[4] Magnus-Alsleben,[5] Parkinson,[6] Price and Mackenzie,[7] Röhmer,[8] and Schwensen[9] have each reported one such case. Korak[10] recently has described two cases of Adams-Stokes disease following diphtheria. However, Jones and White,[11] in a follow-up study of 100 cases of diphtheria over a period of not longer than five to eight years, failed to find heart-block in a single case. It is our belief, however, that diphtheria may influence the onset of heart-block many years after the occurrence of the original infection. We feel that had the 100 cases studied by Jones and

*From the Medical Clinic of the Penter Bent Brigham Hospital, Boston, Massachusetts.

White been followed 20 or 30 years longer, instances of heart-block might have been found. It must be remembered also that, while diphtheria is a relatively common condition, heart-block is rare, and the combined association would necessarily be infrequent. Nor should it be thought that subsequent heart-block need be a common late sequel of diphtheria.

Cases for this study were selected so that only those were included in which the diagnosis of heart-block was proved by electrocardiograms, and in which there was a definite statement in the past history as to whether the patient had or had not had diphtheria in childhood. No cases were included in which this latter question was uncertain. In this regard a recent experience in questioning a patient in the hospital wards is of some interest. The patient was suspected of having heart-block and the routine clinical record gave a negative past history for diphtheria. However, when the direct question was asked by one of us the patient admitted having had severe diphtheria with post-diphtheritic paralysis in childhood and stated that she had not been asked the direct question previously. A positive history, therefore, in our series is definitely positive and comparatively reliable, and some of those routinely reported negative also may have been positive. No patients were included that had been taking digitalis recently. Similarly all cases with a past or present history of coronary occlusion were excluded as well as those suffering from acute rheumatic heart disease. Although it was not intended to include cases having other acute febrile conditions at the time when heart-block was present, none was found. The purpose of this method of choice was to study those cases of heart-block which were otherwise inexplicable.

With these limitations 20 cases taken from the records of the Peter Bent Brigham Hospital and from those seen in private practice were available for study. There were 14 cases of complete heart-block, five of partial block and one which varied from partial to complete block during the period of observation. In all but one the block was permanent. Ten patients had Adams-Stokes syndrome. No case happened to have a syphilitic history or a positive Wassermann reaction.*

The striking fact that came out of this study, as shown by the accompanying table, is that 10 of the 20 cases of inexplicable heart-block or 50 per cent, gave a positive past history of diphtheria. Despite the fact that diphtheria is a common condition, this cannot be an accidental relationship, for a routine questionnaire in 600 consecutive surgical case histories disclosed a positive incidence of diphtheria in only six per cent. There must, therefore, be some element of cause and effect. It is also interesting that the average age of the ten cases that did have

*Since the completion of this report four additional patients having otherwise inexplicable heart-block have been seen by us. Three of them gave a history of having had diphtheria in childhood.

TABLE I

NO.	CASE NO.	SEX	AGE	DIAGNOSIS	VENTRICULAR HEART RATE	BLOOD PRESSURE	WASSERMANN	PAST HISTORY	YEARS SINCE DIPHTHERIA
1.	10,942	f	17	Heart-block, partial. Aortic and mitral insufficiency.	50	120/40	neg.	Diphtheria, severe, at five. Pyelitis at eleven.	11
2.	11,593	f	50	Heart-block, complete. Adams-Stokes disease. Hypertension. Chronic nephritis.	30	190/60	neg.	Diphtheria at eight. Scarlet fever.	42
3.	15,906	f	61	Heart-block, complete. Auricular fibrillation.	40	176/64	neg.	Diphtheria.	50
4.	17,575	m	61	Heart-block, complete. Auricular fibrillation.	30	160/50	neg.	Diphtheria as a child.	50
5.	19,934	m	66	Heart-block, complete. Pernicious anemia. Aortic insufficiency.	40	130/40	neg.	Diphtheria as a child. "Rheumatic gout."	55
6.	19,294	m	50	Heart-block, partial. Chronic arthritis.	60	142/66	neg.	Diphtheria. Pneumonia as a child.	51
7.	22,182	m	32	Heart-block, complete. Adams-Stokes disease.	30	122/55	neg.	Diphtheria, severe, at nine. Asthma at eight.	23
8.	22,720	f	52	Heart-block, complete. Adams-Stokes disease. Cholelithiasis.	30	175/70	neg.	Diphtheria, severe, at six and seven. Paralysis of legs following.	45
9.	25,859	m	28	Heart-block, complete. Ulcer, duodenal.	43	120/60	neg.	Diphtheria, twice. Pneumonia as a child.	18
10.	34,929	f	59	Heart-block, complete. Adams-Stokes disease. Hypertension. Cholecystitis, chronic.	34	190/70	neg.	Diphtheria, severe, at twelve (pulse very slow and feeble, difficulty in walking for one year).	47
11.	9,961	f	64	Heart-block, complete. Adams-Stokes disease. Hypertension.	35	200/122	neg.	Not sure about diphtheria.	
12.	10,061	m	46	Heart-block, partial. Adams-Stokes disease.	35	170/90	neg.	Typhoid only.	
13.	5,458	m	84	Heart-block, partial. Chronic nephritis. Hypertension.	37	220/110	neg.	Negative.	

TABLE I—CONT'D

NO.	CASE NO.	SEX	AGE	DIAGNOSIS	VENTRICULAR HEART RATE	BLOOD PRESSURE	WASSERMANN	PAST HISTORY	YEARS SINCE DIPHTHERIA
14.	15,035	f	72	Heart-block, variable. Adams-Stokes disease. Aortic insufficiency. Hypertension.	40	220/66	neg.	Negative.	
15.	27,035	m	36	Heart-block, complete. Adams-Stokes disease. Congenital heart defect (septum defect).	40	115/60	neg.	Negative.	
16.	28,081	f	65	Heart-block, partial. Adams-Stokes disease. Chronic myocarditis. Hypertension.	45	190/60	neg.	Scarlet fever.	
17.	28,663	m	64	Heart-block, complete. Hypertension.	35	190/70	neg.	"Rheumatism" (no redness and swelling of joints).	
18.	30,786	m	21	Heart-block, complete.	34	124/50	neg.	Infection of foot 6 weeks before, followed by slow heart.	
19.	32,935	m	75	Heart-block, complete. Myocardial insufficiency (congestive). Nephritis, chronic.	30	156/100	neg.	Influenza.	
20.	33,788	m	59	Heart-block, complete. Myocarditis, chronic. Hypertension. Cirrhosis of liver.	35	200/75	neg.	No previous infections.	

SUMMARY

	PATIENTS WITH POSITIVE DIPHTHERIA HISTORY	PATIENTS WITH NEGATIVE DIPHTHERIA HISTORY
Average age, years	47.6 (17 to 66)	58.6 (21 to 84)
Blood pressure, average	153/58 mm.	192/66 mm.
Ventricular heart rate, average	38	37
Average number of years since diphtheria	39 (11 to 55)	

diphtheria was 11 years less than that of the ten that did not. One might infer from this that in the younger patients the previous diphtheria has a greater significance than in the older, and that in the latter group the more customary factor of arteriosclerosis plays the primary rôle. This is well borne out by the fact that the older group without diphtheria had an average systolic blood pressure 40 mm. higher, and an average diastolic pressure 11 mm. higher than those with a past history of diphtheria.

It must be remembered that heart-block itself with the resulting bradycardia and long diastolic pauses of the heart, produces a slight elevation in the systolic and lowering in the diastolic blood pressures. The readings in our cases, therefore, need to be interpreted with this in mind. Thus the group with a positive history of diphtheria, having an average blood pressure of 153/58, may be regarded as having an essentially normal blood pressure level.

Our interpretation of these data is that diphtheria in some way, after a variable latent period, either brings about the impairment in the conduction apparatus as the sole cause of the heart-block, or predisposes the heart to the insidious process of sclerosis which in the absence of diphtheria would have matured at a later age.*

The following three case reports serve to illustrate the type of material with which we are dealing. Two of the patients were young people having normal blood pressures who had complete heart-block with no demonstrable cause except a history of severe diphtheria in childhood. The other was a woman of 52 years characteristic of the older group.

CASE HISTORIES

CASE 7.—Medical No. 22182. A white man, 32 years of age, entered the hospital on October 15, 1923, complaining of slight dyspnea on effort and dizziness for nine months, and in the last two days two attacks of unconsciousness, each lasting about five seconds. His past history revealed "asthma" at eight years and severe diphtheria at nine. His heart rate was 30 and regular. Blood and urine examinations were negative. Blood Wassermann reaction was negative. Vital capacity was 3950 c.c., which was normal. Blood pressure was 122/50 mm. of Hg. Electrocardiograms showed complete heart-block and defective intraventricular conduction. Physical examination showed the heart to be slightly enlarged. A faint systolic murmur was heard at the apex. No diastolic murmur was present. Auricular beats were audible over the precordium and visible in the neck. The diagnosis of Adams-Stokes disease and complete heart-block was made. Vagal pressure, ocular pressure, atropine sulphate gr. 1/30 (0.002 gm.), digitalis folia 2.6 gm. were tried without symptomatic relief or change in the electrocardiographic tracings. Finally barium chloride gr. ½ (0.030 gm.) four times a day by mouth and adrenalin chloride, 1.0 c.c. of a 1/1000 solution subcutaneously 30 minutes after each dose of barium, were given with complete and permanent relief. He has now been entirely free

*A further possibility arises in this connection, i.e., that early diphtheria may be related in a similar causative manner in some of the cases of so-called chronic myocarditis which do not have heart-block hypertension, valvular disease, coronary artery disease and the like.

from symptoms for six years. He took barium chloride for only two weeks. At no time during his stay in the hospital did he have fever.

CASE 8.—Medical No. 22720. A white woman of 52 years was admitted to the hospital December 31, 1923, with the complaints of attacks of fainting and complete unconsciousness increasing in frequency and severity for the last five years. During an attack her pulse could not be obtained and immediately after the attack the rate was 30. She had twice injured herself by falling and on the day before admission had lost control of rectal and bladder sphincters during an attack. She had measles, mumps, whooping cough and scarlet fever as a child. She had diphtheria twice at the ages of six and seven years. Following the second attack there was some paralysis of both legs so that she could not walk for one year. For the last few years she had attacks of pain in the right upper quadrant of the abdomen so severe as to require morphia and strongly suggestive of gall stone colic. The temperature and respiration were normal, the pulse 30 and regular. The blood pressure was 175/70 mm. The blood count and hemoglobin were normal. The Wassermann reaction was negative and she gave no history of syphilis. Kidney function and urine were normal. Electrocardiograms showed complete heart-block with an auricular rate of 50 and a ventricular of 30. The heart was moderately enlarged. The sounds were of good quality. There was a loud harsh systolic murmur over the aortic area but no thrills. No diastolic murmurs were heard. Distinct auricular waves were to be seen in the veins of the neck. During her stay she had three attacks of unconsciousness when no ventricular beat was audible for from 20 to 30 seconds. On the 15th day she began to take barium chloride gr. ½ (0.030 gm.) by mouth three times a day and left the hospital fifteen days later, having had no attacks since the commencement of the barium. The diagnosis was Adams-Stokes disease, complete heart-block and cholelithiasis.

CASE 9.—Medical No. 25859. A white man, 28 years old, entered the hospital on May 14, 1925 complaining of pain in the chest for two years without dyspnea or other evidence of cardiac disease, and epigastric distress typical of peptic ulcer of five years' duration. At the ages of 12 and 13 he had had two severe attacks of diphtheria. His past history was otherwise negative except for measles and whooping cough as a child. His heart was regular with a rate of 43. His blood pressure was 120/60 mm. Electrocardiograms showed complete heart-block, the auricular rate being 63 and the ventricular rate 43. The heart was not enlarged. There were no murmurs except a soft blowing systolic over the base. There was a third sound in some cycles thought to be the auricular beat. The vital capacity was 4150 c.c. or 108 per cent of normal. Blood and urine were entirely negative. He was treated for his ulcer with Sippy management and returned home improved, with the diagnosis of complete heart-block and duodenal ulcer.

SUMMARY

A group of twenty patients having proved heart-block without the customary causes such as coronary artery disease, digitalis, fever and rheumatic infection were studied to determine the incidence of diphtheria in childhood. The incidence was 50 per cent as compared to six per cent in 600 consecutive control surgical cases. The average age of patients with a positive diphtheria history was 11 years younger and their systolic blood pressure 40 mm. lower than was the case with those with a negative history.

Diphtheria in childhood appears to be an etiological factor in the development of heart-block in later years.

REFERENCES

1) Levine, S. A.: The Treatment of the Attacks of Syncope Occurring in Adams-Stokes Disease, Boston Med. & Surg. Jour., **195**, 1147, 1926.
2) Marvin, H. M., and Buckley, R. C.: Complete Heart-Block in Diphtheria, Heart, **11**, 309, 1924.
3) McCulloch, H.: Studies on the Effect of Diphtheria on the Heart, Am. Jour. Dis. Child., **20**, 89, 1920.
4) Fleming, G. B., and Kennedy, A. M.: A Case of Complete Heart-Block in Diphtheria, with an Account of Post-mortem Findings, Heart, **2**, 77, 1910.
5) Magnus-Alsleben: Zur Kenntnis der vorübergehenden Ueberleitungsstorungen des Herzens. Zeitschr. f. klin. Med., **69**, 82, 1910.
6) Parkinson, J.: Auricular Fibrillation Following Complete Heart-Block in Diphtheria, Heart, **6**, 233, 1915.
7) Price, F. W., and Mackenzie, I.: Auricular Fibrillation and Heart-Block in Diphtheria, Heart, **3**, 233, 1911.
8) Röhmer, P.: Elektrocardiographische und anatomische Untersuchungen über den Diphtherieherztod und dessen Beziehungen zum Reizleitungssystem, Zeitschr. f. exper. Pathol. und Therap., **11**, 426, 1912.
9) Schwensen, C.: Et Tilfaelde af komplet Hjertblok som Flge af Difteri, Ugeskrift f. Laeger, **83**, 1395, 1921.
10) Korak, S.: Adams-Stokes Disease in Diphtheria, Med. Klin., **25**, 427, 1929.
11) Jones, T. D., and White, P. D.: The Heart after Severe Diphtheria, Am. Heart Jour., **3**, 190, 1927.

THE DISTRIBUTION OF THE POTENTIAL DIFFERENCES PRODUCED BY THE HEART BEAT WITHIN THE BODY AND AT ITS SURFACE*†

FRANK N. WILSON, M.D.
ANN ARBOR, MICH.

ESSENTIALS OF EINTHOVEN'S TRIANGLE

IN 1913 Einthoven, Fahr, and de Waart[5] made an extremely important contribution to the subject of electrocardiography. They described a method by means of which it is possible to determine the direction and the "manifest value," or in other words the value manifest or effective in the three standard electrocardiographic leads, of the potential difference produced by the heart beat at any given instant in the cardiac cycle.

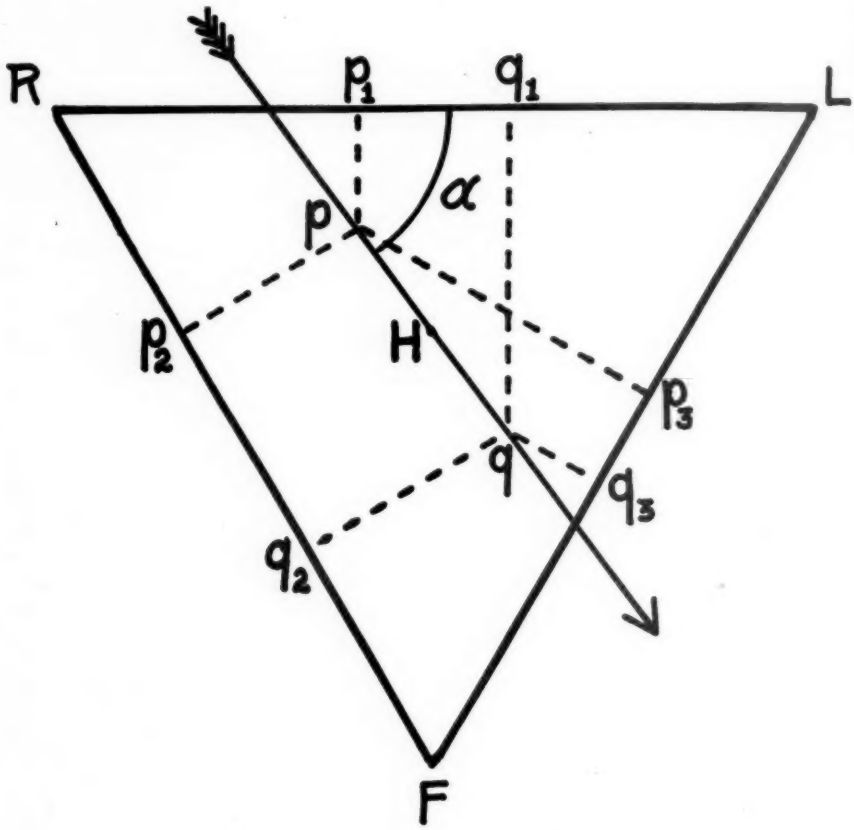
This method is now well known to all students of electrocardiography; nevertheless the principles upon which it is based, and consequently the limitations to which it is subject do not appear to be generally appreciated. Having myself entertained at one time a number of erroneous ideas regarding it, from which I have freed myself only gradually and with much trouble, it seems worth while to give a brief account of my own difficulties in order that they may be avoided by others, and in order to point out that the fundamental principles involved have important applications in many electrocardiographic problems.

Einthoven's original description of the method is a masterly one. All of the assumptions upon which it is based are clearly stated; the method is applied in the solution of several problems, and its use is fully illustrated. It must be remembered, however, that Einthoven was thoroughly familiar with electrical theory and with mathematical physics; he spent most of his life working on problems that required an extensive knowledge of these subjects. Most electrocardiographers, on the other hand, are neither mathematicians nor physicists. Conse-

*From the Department of Internal Medicine, University of Michigan Medical School.

†Most of the observations upon which this article is based were made between 1915 and 1922; some of them were repeated and others were made for the first time between 1922 and 1926. The conclusions incorporated in it date from the same period. A few of the observations and some of the conclusions drawn from them have been published incidentally in papers dealing primarily with other subjects (1, p. 161; 2, p. 237 footnote; 3, p. 101 footnote). In 1926 a preliminary report calling attention to the laws which govern the flow of electric currents in solid conductors, within which a source of potential difference exists, and pointing out that these laws determine the distribution of the potential differences produced by the heart beat within the body and at its surface, was published in collaboration with Wishart and Herrmann[4]. Circumstances beyond the control of the writer have prevented the publication of the complete report until this time; it is now published in order that it may serve as an introduction to further studies based in part upon it which have been carried out recently in collaboration with MacLeod and Barker.

quently, some steps in the development of the method which appeared obvious to Einthoven have not been at all obvious to the majority of those who have made use of it.



$$pq = E$$
$$p_1q_1 = e_1 = E\cos\alpha$$
$$p_2q_2 = e_2 = E\cos(\alpha - 60)$$
$$p_3q_3 = e_3 = E\cos(120 - \alpha)$$

Fig. 1.—After Einthoven, Fahr, and de Waart[5] An equilateral triangle of homogeneous material. A potential difference is assumed to exist between two points very close together near the center of the triangle H. The arrow gives the direction of the potential difference.

In the original article the foundations of the method are described in substance as follows:

Let us assume that the equilateral triangle RLF (Fig. 1) represents a homogeneous flat plate of conducting material, and that a potential difference is produced between two points extremely close together, lying in the immediate neighborhood of its center H. Let us assume further that the line which joins the negative to the positive point has the direction of the arrow drawn through H. Upon this arrow let us lay off an arbitrary distance *pq*, and let the projections of *pq* upon the three sides of the triangle, RL, RF, and LF, be represented by e_1, e_2, and e_3 respectively. Under these conditions the potential difference between any two apices of the triangle must be proportional to the projection of *pq* upon the side of the triangle which joins them; in other words if R-L, R-F, and L-F represent the differences in potential between the corresponding apices, then R-L : R-F : L-F $= e_1 : e_2 : e_3$. Why this must be so is not explained. When the writer asked Einthoven, at the time of his last visit to America, why this explanation was omitted, he replied that it seemed obvious.

It is carefully explained that the manifest potential difference is not to be confused with the actual potential difference at the center of the triangle, of which it is but a small fraction, which varies in value with the distance between the points showing the potential difference. It is pointed out also that when this scheme is applied to the analysis of the human electrocardiogram certain assumptions are made; namely, that the heart is a material point in a homogeneous medium; that the heart is equidistant from the three points to which the electrodes are attached; and that consequently the resistances between these points and the heart are equal. It is admitted that these assumptions are not strictly in accord with the facts. The electrical resistance of the lungs differs from that of the heart and that of the chest wall. The two feet are represented in the triangle by a single point, although actually small but measurable differences of potential between them are produced by the heart beat. The results yielded by the method indicate, however, that these assumptions do not invalidate it for practical purposes.

Einthoven also points out that any potential difference generated in a direction perpendicular to the frontal plane is without influence upon the three standard leads, and that any potential difference which makes an acute angle with this plane exerts an effect proportional to the cosine of this angle.

THE EFFECT OF PLACING ONE ELECTRODE NEAR THE HEART

I first became deeply interested in Einthoven's method of analyzing the form of the electrocardiogram in 1915. I was engaged at that time in a study of atrio-ventricular rhythm. It was observed that in this condition the auricular complex was upright in lead *I* but inverted in leads *II* and *III*. The equilateral triangle seemed to offer

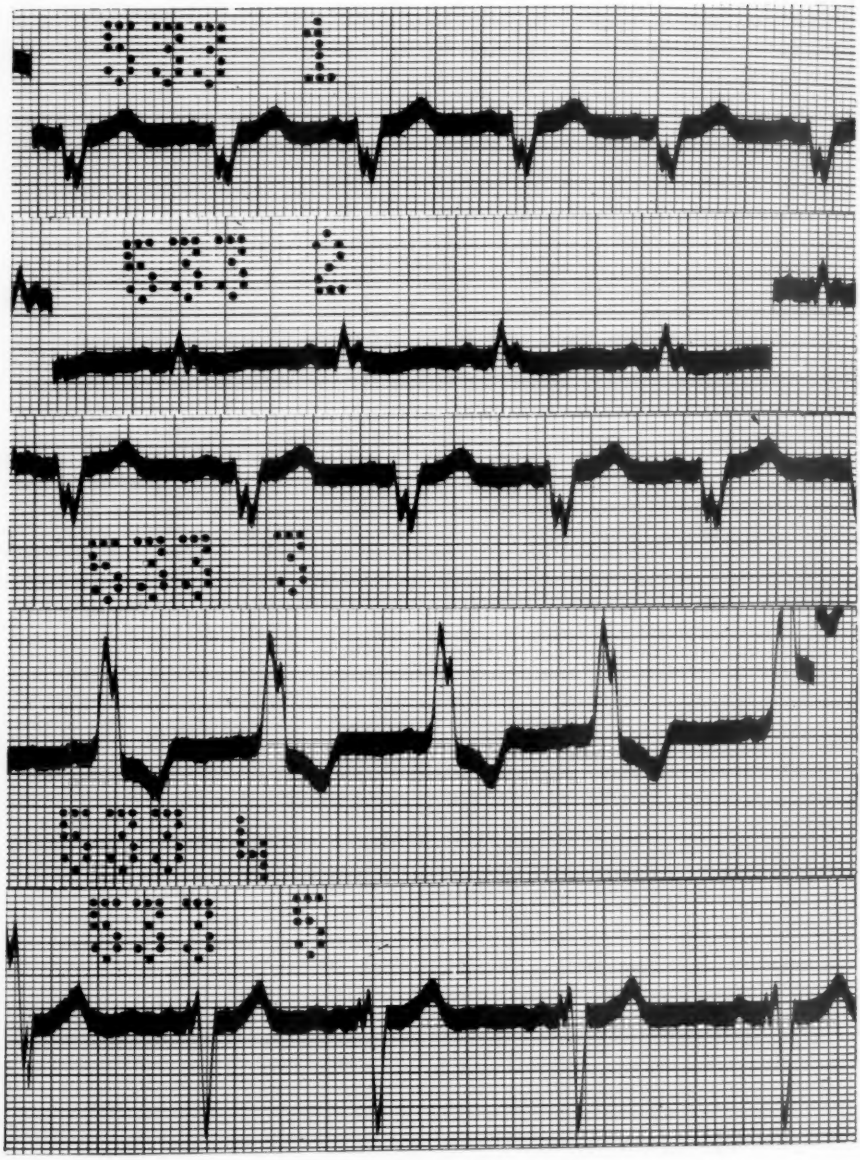


Fig. 2.—Five curves taken by means of chest leads in a case of intraventricular block.

1 cm. equals 1 millivolt, time divisions, 0.2 and 0.04 second.
533-1. *Z*-electrode in 2*sd.* i.c.s. 3.25″ to right of midline.
C-electrode in 2*sd.* i.c.s. 3.25″ to left of midline.
533-2. Same as 533-1 except *C*-electrode moved 2.5″ further to the left.
533-3. Same as 533-1 except *Z*-electrode moved 2.5″ further to the right.
533-4. *Z*-electrode in right axilla, *C*-electrode in left axilla.
533-5. *Z*-electrode just inside right nipple, *C*-electrode just inside left nipple.

an explanation; it indicated that the average direction of the spread of the excitation process in atrio-ventricular rhythm was upward and to the left, a conclusion apparently in harmony with the location of the atrio-ventricular node. Since, however, the triangle gave no information concerning events in the sagittal plane, an attempt was made to make the method three-dimensional by choosing four points on the body surface so arranged as to form the apices of an equilateral tetrahedron, and taking six leads corresponding to the six edges of this figure. At this time the writer was under the impression that since Einthoven appeared to treat the potential difference at the center of the triangle as if it were a mathematical vector, it could be so treated under all circumstances. I supposed that any lead from right to left must give an electrocardiogram similar to, if not identical with, that recorded in lead *I*.

When an attempt was made, however, to substitute an equilateral tetrahedron for the equilateral triangle, it was soon discovered that this was by no means the case. If two leads from right to left are taken; one from the right axilla to the left axilla and another from the right nipple to the left nipple, the chief deflections of the ventricular complex may be upright in one lead and inverted in the other. Fig. 2 illustrates this point; the subject was a patient with intraventricular block. The first curve (533-4) was taken with the *Z*-electrode (right-hand electrode) in the right axilla at the level of the nipple and the *C*-electrode (left-hand electrode) in the left axilla at the same level. Each electrode was then moved 3 in. toward the midline, so that the *Z*-electrode was just inside the right nipple and the *C*-electrode just inside the left. In the first curve the initial ventricular deflections are upright; in the second they are inverted (533-5). A second series of curves was then taken at the level of the second intercostal space. In taking the first of these (533-1) the *Z*-electrode was placed 3.25 in. to the right of the midline and the *C*-electrode the same distance to the left. The *C*-electrode was then moved 2.5 in. further to the left of the midline, the *Z*-electrode being left in place and another curve was taken (533-2). It will be seen that moving the *C*-electrode to the left changed the direction of the chief initial deflection of the ventricular complex. Moving the *Z*-electrode an equal distance to the right of the midline (533-3), the *C*-electrode being returned to its original position, had no appreciable effect. It will be noted also that the curves taken at the level of the second intercostal space are much smaller in amplitude than those taken at the nipple level, although the galvanometer was used at the same sensitivity. These observations suggest that the position of the electrode which is nearest the heart exerts the controlling influence upon the form of the resulting curve. In fact, if one electrode is placed upon the center of the precordium, the position of the second electrode, so long as it is relatively distant from the heart,

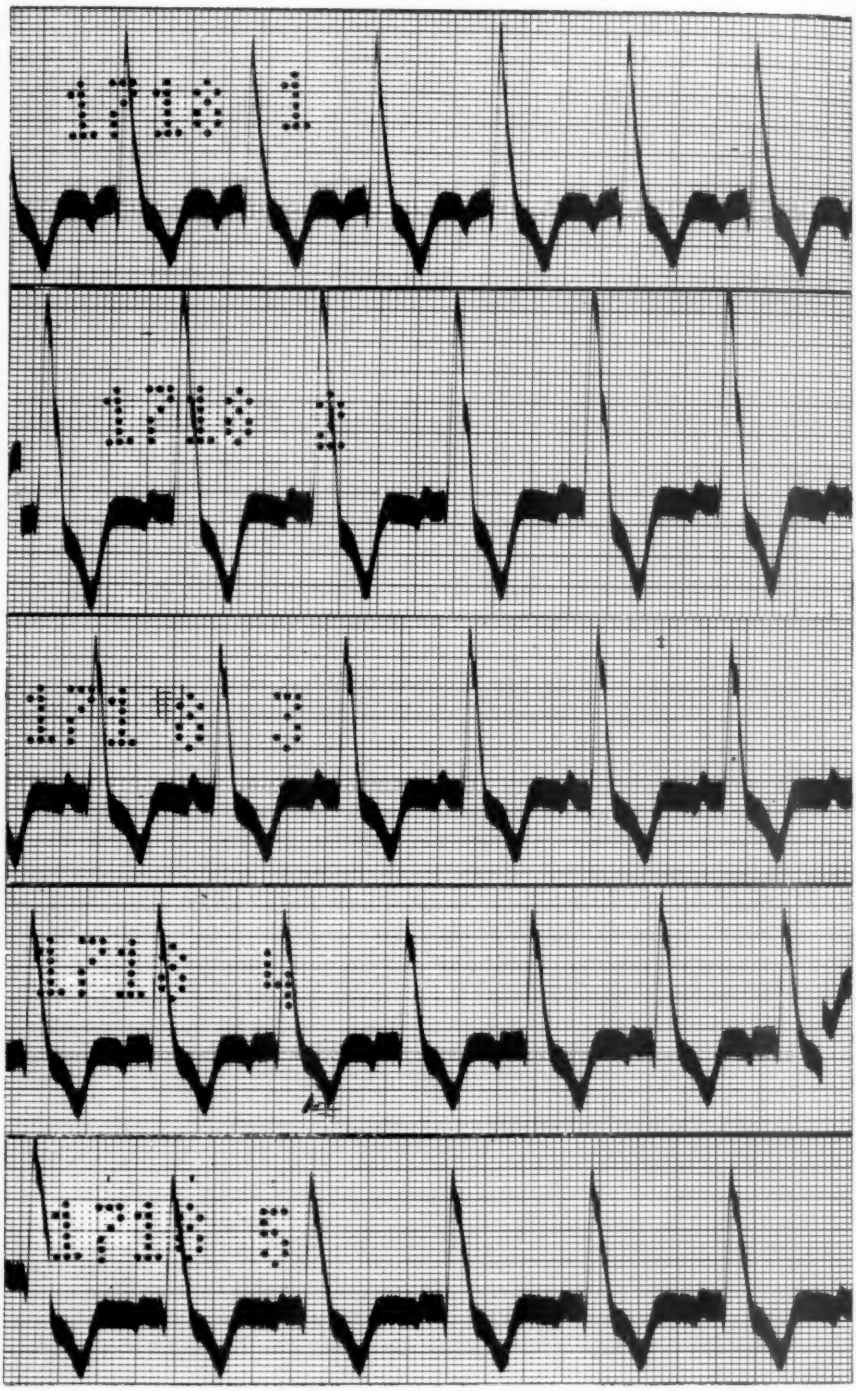


Fig. 3.—Five electrocardiograms taken by means of chest leads. From the same patient as Fig. 2. *Z*-electrode upon 4th costal cartilage near sternum in all instances.

1718-1. *C*-electrode on manubrium.
1718-2. *C*-electrode in left axilla.
1718-3. *C*-electrode on abdomen.
1718-4. *C*-electrode in right axilla.
1718-5. *C*-electrode on spine.

has little effect upon the ventricular electrocardiogram. The five curves shown in Fig. 3 were taken from the same patient as those already described. The *Z*-electrode was placed upon the 4th costal cartilage at its junction with the sternum; the *C*-electrode was placed in turn upon the manubrium (5 in. from the *Z*-electrode; 1718-1); in the left axilla (10 in.; 1718-2); upon the abdomen (9 in.; 1718-3); in the right axilla (8 in.; 1718-4); and upon the spine (1718-5). The ventricular complexes have the same general form in all of these courves although they differ somewhat in amplitude. It appears, therefore, that the potential differences in the immediate neighborhood of the heart are of much greater magnitude than those at a distance from it. This may be demonstrated most easily by the following experiment. Let a line be drawn from the 4th costal cartilage near the sternum to any point on the anterior aspect of the left thigh. Starting at the proximal end of this line (4th costal cartilage) divide it into equal segments five inches in length. From each of these segments take an electrocardiogram, placing the *Z*-electrode upon the proximal end (end toward the heart) and the *C*-electrode upon the distal end of the segment. A series of curves taken in this manner has been published by Wilson and Herrmann.[1] The first segment yields a curve of large amplitude; in succeeding segments the curves become rapidly smaller and from the fourth or fifth and the remaining segments no curves at all are obtained, providing the sensitivity of the galvanometer is not increased. In a similar way it may be shown that, with the galvanometer at normal sensitivity, all points upon the left leg have the same potential throughout the cardiac cycle and the same is true of all points on the left arm, and of all points on the right arm. As Einthoven pointed out points on the right leg differ only very slightly in potential from points on the left.

Einthoven's reasons for pointing out that the equilateral triangle is based upon the assumption that the three apices of the triangle are equidistant from the heart are now obvious. It is also apparent that the three standard leads have a very great advantage over any other similar system of leads that could be employed. Since all points on the right arm, for instance, have the same potential, when the galvanometer is employed at the standard sensitivity, it does not matter whether the electrode connected to this arm is placed upon the wrist, the hand, the forearm, or the upper arm; the arm acts merely as an extension of the wire attached to it; the lead is from the attachment of the arm to the trunk. The same is true of the left arm and of the left leg. If the electrodes are placed upon the trunk, however, the exact position of each electrode, particularly if it be less than 10 or 15 inches from the heart is a matter of importance. Obviously, if three points on the trunk which form the apices of an equilateral triangle are substituted for the right arm, left arm, and left leg, and

three leads corresponding to the three standard leads are employed, the resulting electrocardiograms may be analyzed by the method of the equilateral triangle, but no confidence can be placed in the results unless it can be demonstrated that the three points are equidistant from all parts of the heart, or that they are so far from the heart, that any difference between them in this respect is of no importance. The writer is of the opinion that the principles of the equilateral triangle should not be applied to any system of chest leads whatsoever, particularly if it is desired to analyze the ventricular complex. Since it is practically impossible to choose three points in the sagittal plane which form the apices of an equilateral triangle, and which, at the same time, are both distant and equidistant from the heart, it does not seem to the writer that the principles of the equilateral triangle can justifiably be employed in studying the potential differences which have an antero-posterior direction, except perhaps in the case of the auricular deflections where the error introduced by placing one electrode nearer the heart than the other appears to be less than in the case of the ventricular deflections.

Observations such as those described convinced the writer some ten years ago that he could make no progress in the analysis of the electrocardiograms taken by means of chest leads, or by placing one or both electrodes upon the surface of the exposed heart, until he understood the laws which govern the distribution of potential differences within solid conductors. A careful examination of the older electrocardiographic literature did not disclose any discussion of these laws, although it was evident that they were known to Waller as early as 1889. In a paper which appeared in that year[6] he published an outline drawing of the trunk upon which a system of iso-potential surfaces, seen in cross-section, are drawn about the heart. Waller was under the impression that the impulse spread over the ventricular muscle from apex to base in the form of a peristaltic wave; and that the muscle of the ventricles could be treated as a single unit. Consequently, he represented the iso-potential surfaces as if the apex of the heart could be treated as the negative pole or sink and the base as the positive pole or source of the potential differences produced by the ventricles. Waller used this diagram in explaining why some leads gave larger deflections than others. He did not discuss the laws upon which the diagram is based. These laws were obviously known to Einthoven also, since the equilateral triangle is founded upon them; his discussion of the assumptions upon which the application of the principles of the triangle is based shows plainly that, as might be expected from his knowledge of electrical theory, he knew and understood the factors involved thoroughly. He did not, however, discuss the laws themselves, which in so far as they apply to simple conditions can be found in almost any textbook dealing with electrical theory or with

potential function. The distribution of potential differences in a solid conductor so irregular in shape as the body cannot, of course, be determined mathematically. Nevertheless, the laws as they apply to homogeneous solids of regular shape or of infinite extent are very helpful in understanding the principles involved.

LAWS WHICH GOVERN THE DISTRIBUTION OF POTENTIAL DIFFERENCES

The potential, V, of any point in a thin homogeneous sheet of conducting material, infinite in extent, within which a potential difference

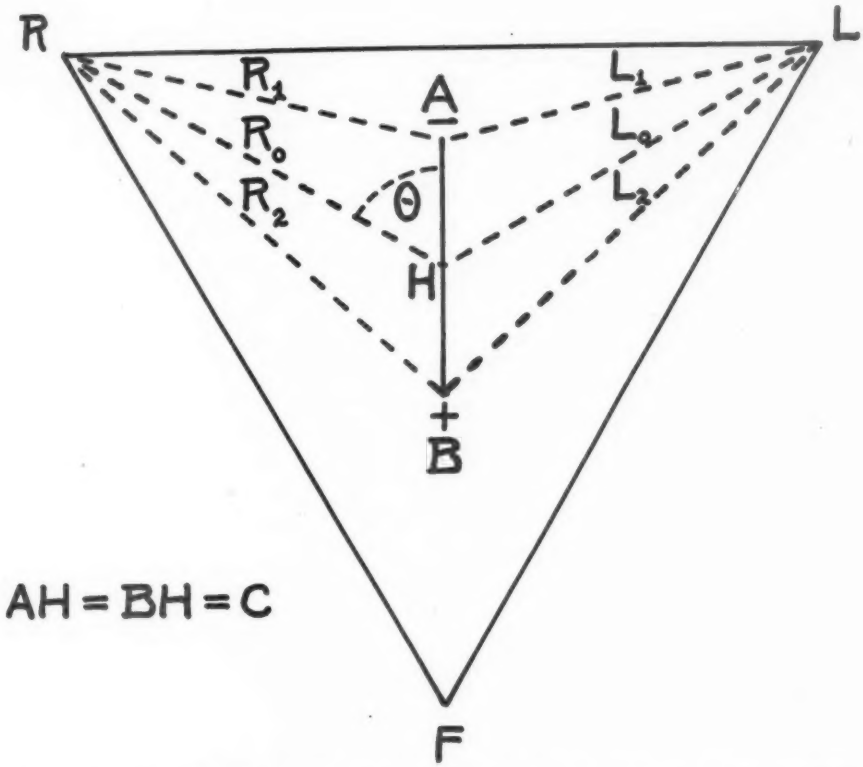


Fig. 4.—An equilateral triangle in an infinite conducting sheet or infinite conducting solid; a potential difference between A and B which are equidistant from the center H. The source of this potential difference at B (+) and the sink at A (−). C is equal to one-half the distance between sink and source. Line AB is the axis of the potential difference.

is maintained between two points close together is determined by the equation below[7];—

$$V = \frac{Q}{2\pi kd} \log_e \frac{R_1}{R_2} \qquad (1)$$

In this equation Q is the quantity of electricity flowing in unit time; k, the conductivity of the material of which the sheet is composed; d, the thickness of the sheet; and R_1 and R_2, the distances of the

point from the negative pole or sink and the positive pole or source of the potential difference respectively.

Let us construct (Fig. 4) upon this infinite sheet of conducting material an equilateral triangle RLF so placed that the sink and source of the potential difference will be near to and equidistant from the center of the triangle H. The difference in potential between two apices of the triangle, R and L, will then be determined by the following expression.

$$V_1 - V_2 = \frac{Q}{2\pi dk} \log_e \frac{R_1}{R_2} - \log_e \frac{L_1}{L_2}) \qquad (2)$$

In this expression V_1 is the potential of apex R; V_2 the potential of apex L; R_1 and R_2 the distances of R_1 and L_1 and L_2 the distances of L from sink and source respectively.

It will be seen at once that if the line joining these two apices of the triangle (RL) is perpendicular to the axis of the potential difference, that is to say to the line joining sink and source, both apices will have the same potential, for the ratio $\frac{R_1}{R_2}$ will be equal to the ratio $\frac{L_1}{L_2}$. It is also clear that the difference in potential between R and L will be maximal when the line joining them is parallel to the axis of the potential difference. It is not quite so obvious that if the sink and source are very close together the potential difference between R and L will be proportional to the cosine of the angle between the axis of the potential difference and the line RL. This may be shown as follows:

The potential V_1 of the apex R is determined by $\log_e \frac{R_1}{R_2}$. This expression may be represented by an infinite series (9); viz.,—

$$\log_e \frac{R_1}{R_2} = 2 \left[\frac{R_1 - R_2}{R_1 + R_2} + \tfrac{1}{3} \left(\frac{R_1 - R_2}{R_1 + R_2} \right)^3 \ldots\ldots \text{etc.} \right] \qquad (3)$$

$$= 2 \left[\frac{R_1^2 - R_2^2}{(R_1 + R_2)^2} + \tfrac{1}{3} \left(\frac{R_1^2 - R_2^2}{[R_1 + R_2]^2} \right)^3 \ldots\ldots \text{etc.} \right] \qquad (4)$$

Referring again to Fig. 4, it will be seen that R_1^2 is equal to $R_0^2 + C^2 - 2R_0C \cos \theta$ when R_0 is the distance of R from the center of the triangle and C is one-half the distance between sink and source. Similarly, R_2^2 equals $R_0^2 + C^2 + 2R_0C \cos \theta$.

If we substitute these values of R_1^2 and R_2^2 in equation (4) we get

$$\log_e \frac{R_1}{R_2} = 2 \left[\frac{-4\, R_0C \cos \theta}{(R_1 + R_2)^2} + \tfrac{1}{3} \left(\frac{-4\, R_0C \cos \theta}{(R_1 + R_2)^2} \right)^3 \ldots\ldots \text{etc.} \right] \qquad (5)$$

If sink and source are extremely close together R_1 and R_2 will become practically equal to R_0 and this expression will become

$$\log_e \frac{R_1}{R_2} = 2\left[\frac{-C\cos\theta}{R_0} + \tfrac{1}{3}\left(\frac{-C\cos\theta}{R_0}\right)^3 \ldots\ldots \text{etc.}\right] \qquad (6)$$

This series is rapidly convergent and since R_0 is very large we may neglect all terms except the first. In that case

$$\log_e \frac{R_1}{R_2} = \frac{-2\,C\cos\theta}{R_0}. \quad \text{Hence } V_1 = \frac{Q}{2\pi dk}\left(\frac{-2C\cos\theta}{R_0}\right) \qquad (7)$$

Since $\angle$RHL equals 120° and R_0 equals L_0, it may be shown in the same way that, if V_2 is the potential of L

$$V_2 = \frac{Q}{2\pi dk}\left[\frac{-2\,C\cos(120^\circ - \theta)}{R_0}\right] \qquad (8)$$

$$\text{Consequently, } V_1 - V_2 = \frac{Q}{2\pi dk}\left[\frac{-2\,C}{R_0}\right]\left[\cos\theta - \cos(120^\circ - \theta)\right] \qquad (9)$$

But θ may be expressed in terms of $\propto$, the angle between the axis of the potential difference and the line RL for as may be seen from Fig. 4, $\theta = \propto - 30^\circ$. Therefore,

$$V_1 - V_2 = \frac{Q}{2\pi dk}\left[\frac{-2\,C}{R_0}\right]\left[\cos(\propto - 30^\circ) - \cos(150^\circ - \propto)\right] \qquad (10)$$

$$V_1 - V_2 = \frac{Q}{2\pi dk}\left[\frac{-2\,C}{R_0}\right]\left[\sqrt{3}\cos\propto\right] \qquad (11)$$

Q.E.D.

The expression for the potential of any point in a homogeneous conductor of which all the dimensions are infinite is even more simple[8] than that which holds for an infinite thin sheet. It is

$$V = K\left(\frac{1}{R_2} - \frac{1}{R_1}\right) \qquad (12)$$

In this expression K is a constant depending upon the conductivity of the medium and the quantity of electricity flowing in unit time, and R_1 and R_2 are the distances of the point from the sink and source respectively.

In this case also it may be shown that if an equilateral triangle is so placed in the medium that its apices lie in the same plane as the sink and source, and the sink and source are very close together and

equidistant from the center of the triangle, the difference in potential between any two apices of the triangle will be proportional to the cosine of the angle between the line joining them and the axis of the potential difference at the center. For, referring again to Fig. 4,

$$V_1 = K\left(\frac{1}{R_2} - \frac{1}{R_1}\right) = K\left(\frac{R_1 - R_2}{R_1R_2}\right) = K\left[\frac{R_1^2 - R_2^2}{R_1R_2(R_1 + R_2)}\right] \quad (13)$$

If in this expression we substitute for R_1^2 and R_2^2 their values in terms of R, C and θ we get,

$$V_1 = K\left[\frac{-4\,R_0C\cos\theta}{R_1R_2(R_1 + R_2)}\right] \quad (14)$$

When the sink and source are extremely close together so that R_1 and R_2 become practically equal to R_0 this expression becomes

$$V_1 = K\left[\frac{-2\,C\cos\theta}{R_0^2}\right] \quad (15)$$

The remainder of the proof is the same as in the case of an infinite sheet.

In case the sink and source do not lie in the same plane as the triangle so that the line joining them makes an angle with this plane, the effect of the potential difference between them in this plane will be proportional to the cosine of this angle.

It will be observed that the potential difference between any two apices of the triangle (Fig. 4) varies directly with C and consequently it will become greater as the sink and source are further apart. Furthermore since the potential difference varies inversely with R_0 or with R_0^2 according to whether we are dealing with a thin sheet or an infinite solid, the difference in potential will become smaller as R_0 increases. Einthoven's reasons for stating that the manifest potential difference is only a small fraction of the actual potential difference, and that this fraction varies with the distance between the points showing the potential difference, are now obvious.

ILLUSTRATIVE EXPERIMENTS

In order to illustrate the effect of some of the factors involved the writer, in 1922, performed some experiments upon a model. A large shallow pan upon the bottom of which a large equilateral triangle had been drawn was partially filled with weak copper sulphate solution. The three lead wires of the galvanometer were attached to electrodes placed at the apices of the triangle. At the center of the triangle and equidistant from it two other electrodes were placed and these were

connected to the terminals of the secondary coil of an inductorium. The primary circuit of this inductorium was opened and closed rhythmically by a device which short-circuited the secondary coil at the time when the primary circuit was closed so that only break shocks were delivered to the electrodes at the center of the triangle. The effect of these shocks upon the differences in potential between the apices of the triangle was recorded with the string galvanometer. A summary of these experiments is given in Table I.

It will be seen that the deflections in the three leads are proportional, or nearly proportional, to the cosine of the angle between the

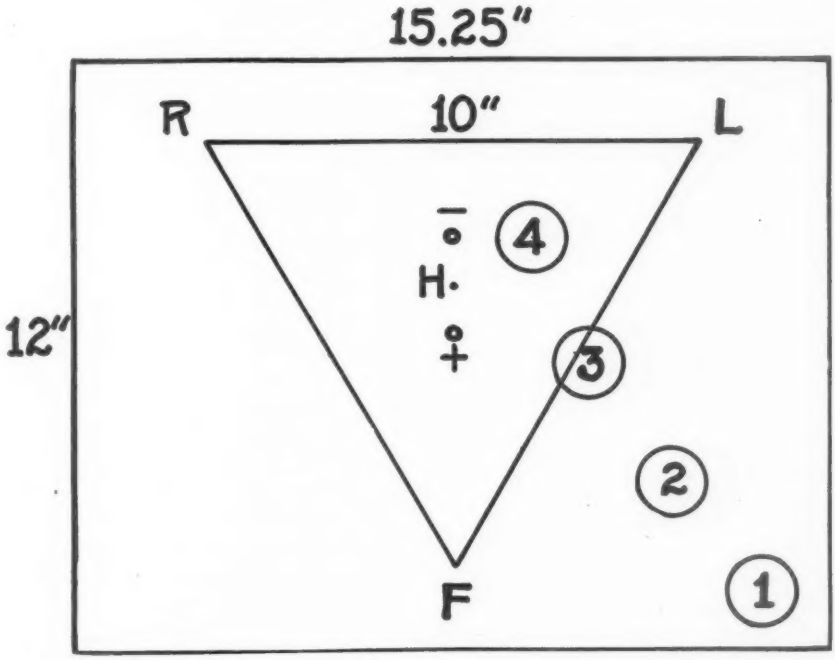


Fig. 5.—An equilateral triangle drawn upon the bottom of a large flat dish containing a weak solution of copper sulphate. The three usual lead wires were attached to the apices of the triangle. Rhythmic induction shocks were delivered to the electrodes near the center H. The circles show the positions of a coil of wire in an experiment described in the text.

line of lead and the axis of the potential difference so long as the sink and source are equidistant from the center of the triangle. If, however, the sink and source are moved to the right or left so that the midpoint of the line which joins them is no longer equidistant from the apices of the triangle this is no longer the case, the direction of the deflection in any lead being then largely determined by the relative distance from the sink and source of the apex which is nearest this point. It will also be observed that in accordance with the laws which describe the flow of currents in solid conductors the manifest potential difference increases with the distance between sink and source.

TABLE I

NO. OF OBSERVATIONS	LENGTH OF SIDE OF TRIANGLE	C**	α††	LI	LII	LIII
1	9.6″	0.65″	30°	14	15	1
2	9.6″	1.00″	30°	17	19	2
3	9.6″	1.50″	30°	26	27.5	1
4	9.6″	1.25″	0°	17	8	–9
5	10.5″	0.68″	90°	–2	7	11
6*	10.5″	0.68″	90°	–5	6	10
7†	10.5″	0.68″	90°	1	7	7
8‡	10.5″	0.68″	90°	4.5	8	3.5

*Both electrodes moved 1.25″ to right (i.e. toward side RF in Fig. 4).
†Both electrodes moved 1.25″ to left (toward side LF in Fig. 4).
‡Both electrodes moved 2.5″ to right and 1.5″ upward.
**C is one half the distance between sink and source.
††α is the angle between the line joining sink and source and line RL (Fig. 4).

In order to determine the effect of changing the resistance between two apices of the triangle upon the difference in potential between them a coil of copper wire was placed in the copper sulphate solution. The various positions of the coil are shown in Fig. 5. The corresponding deflections in lead *III* were as follows; coil out, 18; coil at 1, 16; coil at 2, 13; coil at 3, 9; coil at 4, 6 mm. It will be seen, therefore, that as the coil was moved toward the source of potential difference the magnitude of the deflection in lead *III* rapidly decreased until it reached one-third its value with the coil out.

APPLICATIONS TO DIRECT AND SEMI-DIRECT LEADS

We may now discuss briefly the application of the principles which govern the distribution of potential differences in solid conductors to the analysis of electrocardiograms obtained by direct leads or by indirect leads in which one electrode is placed nearer the heart than the other.

As Lewis[10] has pointed out, the heart muscle cannot be regarded, from the electrocardiographic standpoint, as a single unit; it is obviously made up of a large number of individual units, each of which produces its own electrical effects. Consequently, many sinks and sources must exist within the heart throughout the period of its electrical activity. When the electrodes are placed upon points distant from the heart, all of the muscle units will be equal or practically equal, with respect to their distance from the electrodes and all parts of the heart will have an equal opportunity to exert their influence upon the form of the electrocardiogram. When, on the other hand, one electrode is placed much nearer to the heart than the other, this is no longer the case. The potential variations of the electrode which is placed close to the heart will not only be very much greater than those of the distant electrode, but they will represent the activity of various portions of the heart unequally. Those portions of the heart which are nearest to the electrode which is near by must exert a very much greater effect in proportion to the potential differences which they

produce than those parts of the heart which are further away. Lewis[10] and his associates have shown that when one or both electrodes are placed directly upon the exposed heart, two types of effects can be distinguished; intrinsic effects which are the result of the activity of the muscle immediately beneath the electrode or electrodes placed upon the heart muscle, and extrinsic effects produced by the activity of muscle that is at a distance. It is obvious, however, that an electrode which is placed upon the heart bears no special relation to the subjacent muscle except that of nearness, and that there can be no fundamental difference between placing an electrode actually upon the muscle and placing an electrode close to it, provided of course that in the second case the electrode is not separated from the muscle by a non-conducting substance.

When one electrode is placed upon the precordium and the other at a point relatively distant from the heart, the precordial electrode is much nearer the anterior wall of the heart than the posterior; consequently the electrical activity of the anterior wall of the heart has a much greater effect upon the form of the curve than the electrical activity of the posterior wall, just as the subjacent muscle exerts a more pronounced effect than the more distant muscle in the case of direct leads. Leads in which one electrode is placed close to the heart are therefore semi-direct leads. It is not surprising that the curves obtained from such leads are in many respects similar to those obtained by placing one electrode upon the exposed heart. In both cases the position of the second electrode, so long as it is placed upon a point distant from the heart, has comparatively little effect upon the form of the curve recorded. When one electrode is placed upon the ventricular surface and the other upon a distant point, the arm or leg for instance, deflections are obtained which have a value of 40 to 80 millivolts, a value approximately 20 times that of the tallest deflections that occur in the standard leads. Since the arm and leg do not show a difference of potential exceeding three or four millivolts at any time during the cardiac cycle, which extremity is used as the distant point is relatively immaterial. Consequently, when one electrode is placed upon the heart and the other upon one of the extremities the resulting curve is, for all practical purposes, a record of the variations in potential of the electrode placed upon the heart. The potential variations of a point upon the precordium are very much smaller than the potential variations of a point upon the heart; they are still five to ten times as great as the potential variations which occur at points upon the extremities.

Let us now examine the curves which are obtained by placing one or both electrodes upon the auricular muscle where the course of the excitation wave is a relatively simple one. Lewis[10] and his collaborators found that if the Z-electrode was placed upon the sinus node and

the *C*-electrode a short distance from it the first deflection of the resulting electrogram began with a sharp upright deflection, the intrinsic deflection due to activation of the muscle beneath the *Z*-electrode. If now the *Z*-electrode was placed a short distance from the node and the *C*-electrode still further away the electrogram still showed a sharp upstroke signaling the arrival of the excitation process at the *Z*-electrode, but in this case the intrinsic deflection was preceded by a small deflection downward due to muscle activity at a distance; an extrinsic effect. Obviously, however, this extrinsic effect must be the result of the activation of the muscle immediately about the sinus node for no other muscle is active at the time when it occurs. All of the potential differences which exist under these circumstances must be closer to the *Z*-electrode than to the *C*-electrode. Since the extrinsic deflection indicates relative positivity of the *Z*-electrode this electrode must lie nearer to the positive pole or poles or further from the negative pole or poles than the *C*-electrode. Since, however, all of the active muscle is nearer the *Z*-electrode than the *C*-electrode the second alternative is impossible. Consequently it must be concluded that the positive pole lies between the *Z*-electrode and the active muscle, which is known to show relative negativity. The excitation process must therefore consist in a negative and a positive pole lying relatively close together. Were it not so it would be difficult or impossible to distinguish between intrinsic and extrinsic effects. If, for instance, the excitation process be regarded as a wave of negativity, the whole of the unexcited muscle being regarded as the positive pole, the *Z*-electrode should become more and more negative as the excitation wave approached it; it could never become relatively positive until the wave of negativity had passed the midpoint between the two electrodes, so that the negative pole of the potential difference was closer to the *C*-electrode than to the *Z*-electrode. Under these circumstances there could be no sharp upstroke marking the arrival of the excitation process at the electrode nearest the sinus node, except in the case where this electrode was placed upon the nodal region.

THE CONDUCTIVITY OF THE BODY TISSUES

According to equation (2) the potential difference between apices R and L of the equilateral triangle (Fig. 4) varies inversely with the electrical conductivity of the material within which the source of potential difference exists. We may predict, therefore, that any increase in the conductivity of the body tissues, particularly of those tissues which lie in close proximity to the heart, will decrease the amplitude of the electrocardiographic deflections.

It is well known that in many cases of advanced cardiac disease electrocardiograms of very small amplitude occur. It is generally believed that such curves are the result of a decrease in magnitude of

the potential differences produced by the diseased heart muscle. Admitting that in many, if not in most instances, this is the most reasonable explanation of curves of small amplitude, we may point out that an increase in the conductivity of the body tissues, particularly of those tissues which lie close to the heart, may produce the same result. The lungs because of the large amount of air which they contain must, as Einthoven pointed out, have a somewhat lower electrical conductivity than the chest wall or the heart. The question arises, therefore, as to whether edema of the lungs, pericardial effusion, pleural effusion, hydrothorax, ascites, or massive edema of all the body tissues may not decrease the amplitude of the electrocardiographic deflections.

With this in mind I examined a small group of unselected cases in which electrocardiograms of small amplitude had been recorded. In these cases all of the electrocardiographic deflections were small in all leads; the QRS deflection of largest amplitude did not exceed 0.6 millivolt in value. Of the 24 cases studied, 14 showed advanced cardiac failure with pronounced edema, often associated with hydrothorax or ascites. There were 2 cases of cardiac failure with slight edema and 2 of cardiac failure without evident edema at the time of the examination. There was one case of Hodgkin's disease with massive pleural effusion, and one case of pulmonary tuberculosis and aortic regurgitation with ascites, but without signs of cardiac failure. The clinical diagnoses in the remaining four cases were as follows; arteriosclerosis without cardiac failure, Addison's disease, diabetes mellitus, and pernicious anemia. None of these last patients had cardiac failure, edema, ascites, or other accumulations of fluid at the time of the examination, although the patient with pernicious anemia gave a history of very recent pronounced edema.

In those cases in which cardiac failure and edema were both present, it is, of course, impossible to say whether the condition of the heart muscle or the presence of edema was responsible for the small amplitude of the electrocardiographic deflections. There seems, however, to be a tendency for small curves to occur in patients with edema, ascites, or pleural effusion who have no cardiac failure. In these it is possible that a change in the conductivity of the body tissues is the cause of the small curves. In still other cases the small amplitude of the electrocardiograms is very probably more or less accidental, and is due to the fact that the potential differences produced by one part of the heart are almost exactly neutralized by those produced in other parts, so that the resultant potential difference is very small. This is undoubtedly the explanation in those cases where only part of the electrocardiographic deflections are small; that is to say when only the ventricular or only the auricular complex is affected. Changes in the conductivity of the body tissues must affect the amplitude of all of the

deflections equally, and the same is probably true to a lesser extent of changes in the condition of the heart muscle.

SUMMARY

The foundations of Einthoven's equilateral triangle are discussed and it is pointed out that it is based upon certain assumptions to which Einthoven called particular attention, but the importance of which has not always been borne in mind.

The laws which govern the distribution of potential in solid conductors are described, and it is pointed out that a knowledge of these laws is essential to the analysis of those electrocardiograms obtained by chest leads in which one electrode is placed nearer the heart than the other, and in the analysis of the curves obtained by direct leads.

Leads in which one electrode is placed upon the precordium and the other at a distant point are semi-direct leads. In such leads the electrical effects of that part of the heart wall nearest the precordial electrode are exaggerated.

The excitation wave cannot be regarded as a wave of negativity, since the positive pole of the potential difference which it produces is close to the negative pole. It is this fact which makes it possible to distinguish between intrinsic and extrinsic effects in direct leads.

An increase in the conductivity of the body tissues, particularly of those which are close to the heart must decrease the amplitude of the electrocardiographic deflections. It is suggested that in certain instances accumulations of fluid near the heart or massive edema may act in this way.

REFERENCES

1. Wilson and Herrmann: Arch. Int. Med., **16**: 153, 1920.
2. Wilson and Herrmann: Heart, **8**: 229, 1921.
3. Herrmann and Wilson: Heart, **9**: 91, 1922.
4. Wilson, Wishart, and Herrmann: Proc. Soc. Exper. Biol. and Med., **23**: 276, 1926.
5. Einthoven, Fahr, and de Waart: Arch. f. d. ges. Physiol. **150**: 275, 1913.
6. Waller: Phil. Trans. Roy. Soc., B, **180**: 169, 1889.
7. Electrokinetics, Ency. Brit., 11th ed., **9**: 216.
8. Pierce: Newtonian Potential Function, 3rd Ed., Ginn and Co., p. 248, 1902.
9. Granville: Differential and Integral Calculus, Ginn and Co., p. 236, 1911.
10. Lewis: The Mechanism and Graphic Registration of the Heart Beat. 3rd Ed., Shaw and Sons, London, p. 104, 1925.

PARTIAL BUNDLE-BRANCH BLOCK
A CASE OF THREE-TO-ONE AND FOUR-TO-ONE BLOCK*

SOLOMON R. SLATER, M.D.
BROOKLYN, N. Y.

A RECORD was obtained in the electrocardiographic laboratory which presented such interesting features that its report has seemed warranted. Furthermore, a very extensive search into the literature has failed to reveal a similar electrocardiogram. It is the belief of the writer that it is the first of its kind on record. It was interpreted as partial bundle-branch block in which there was a three-to-one and four-to-one block of the right branch of the bundle of His.

The patient presented herself to the electrocardiographic department on the afternoon of January 8, 1930, at the request of the Thyroid Clinic of the Jewish Hospital. Only a short strip was taken—Fig. 1 representing its entire length. Because of its interest other records were taken that evening, at the same time certain tests being made. As a result of the ergotamine tartrate (gynergen) which she received she began to vomit, felt weak and had to be admitted to the hospital on the service of Dr. Joseph Rosenthal. The following history was obtained and physical findings noted:

G. G., aged forty years, born in Russia was admitted the evening of January 8, 1930 with a story of vomiting, palpitation and dyspnea for the past six weeks. She was short of breath and complained of marked palpitation and progressive weakness. She had lost 25 pounds in six weeks. She was also troubled by a choking sensation and difficulty in swallowing.

On physical examination there was presented a very thin short slightly built female adult who looked much older than her given age. There was a moderately fine tremor of the tongue and an acetone odor to her breath. Pigmentation of the skin of the face and neck was noted. The isthmus of the thyroid gland was palpable. Marked tachycardia was present on admission and the limits of heart dulness were increased. There was a soft systolic murmur present at the apex. These latter findings disappeared. There was impairment of resonance at the right apex and an occasional râle, crepitant in nature, was present after coughing. She had never received digitalis before admission.

Laboratory Data.—The blood study showed a secondary anemia; 4,000,000 erythrocytes and 45 per cent hemoglobin. The blood chemistry was normal and the Wassermann reaction was negative. The urine showed a faint trace of albumin, no sugar and a marked acetone reaction. The sputum examinations were all negative. The basal metabolism showed a plus 30 per cent rate. The x-ray plate of the lungs showed a bilateral hilum infiltration. The blood pressure was 140/70 to 130/50 mm. Hg. The teleroentgenogram of the heart read "Cardiac shadow is within normal limits of size, shape and position." The diagnosis therefor was acute hyperthyroidism with myocardial involvement.

*From the Department of Cardiology, Jewish Hospital of Brooklyn, N. Y.

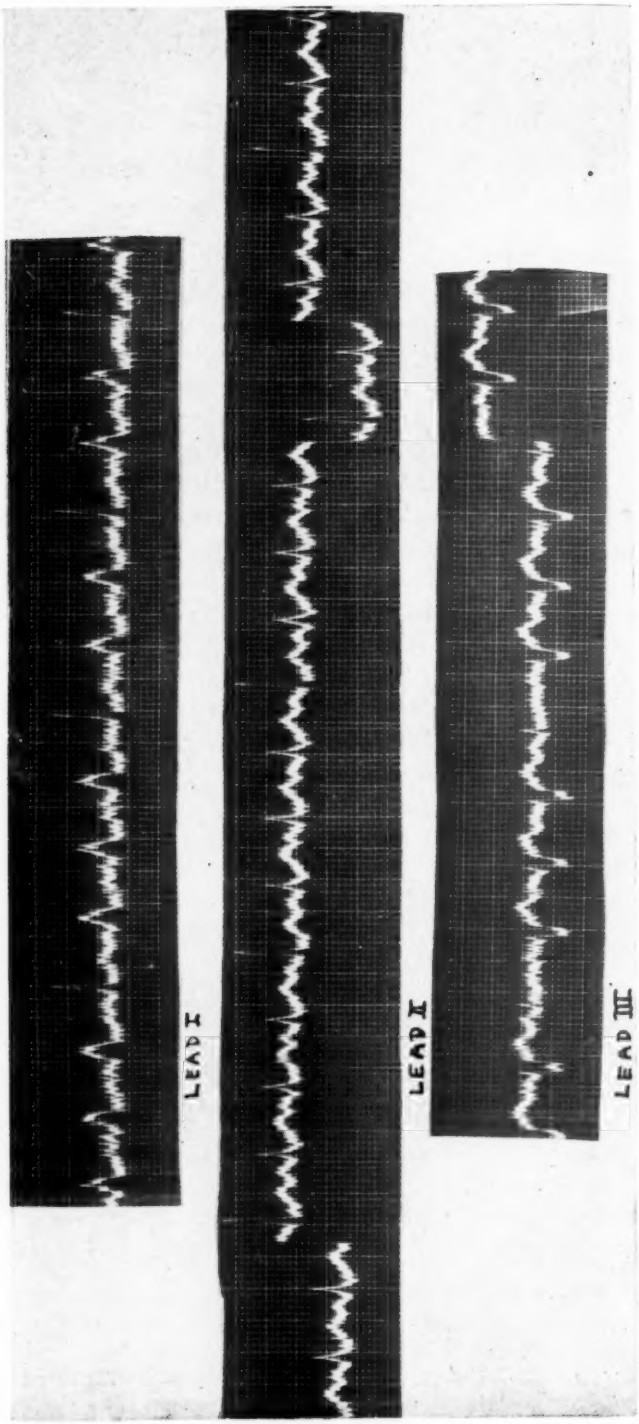


Fig. 1.

DISCUSSION OF THE ELECTROCARDIOGRAMS

The first electrocardiogram was taken on the afternoon of January 8, 1930. (Fig. 1.)

The examination of lead one shows the first three complexes are wide, slurred and notched with an oppositely directed T-wave coming off directly from the downlimb of the R. The first two complexes are distorted somewhat due to irregularity in the running of the camera. The QRS measures 0.12 second. The fourth complex is normal in contour and the T is directed upward. The QRS of this complex measures 0.06 second. This latter complex is followed by three complexes similar to the first three, and the eighth complex is again of normal configuration. The ninth and tenth complexes are wide and broad like the first three and are followed by another normal complex. Again two more abnormal complexes, which are followed by another normal complex. The P cannot be definitely determined in this lead. Because of the somatic tremor (the patient had acute hyperthyroidism), measurements may not be exactly accurate although they were made on many different occasions until absolute correspondence was obtained. These measurements for lead one are as follows: the ventricular rate is 140.8 per minute; the P-R interval could not be measured in this lead; the QRS of the abnormal complexes measured 0.12 second and that of the normal complex 0.06 second. R-R intervals are as follows:

R-R	1.	.410?	R-R	8.	.415
	2.	.450?		9.	.425
	3.	.440		10.	.425
	4.	.410		11.	.410
	5.	.440		12.	.425
	6.	.425		13.	.435
	7.	.435		14.	.425

In lead two, the first two complexes are short and widened, so that the QRS of each of these complexes measures 0.12 second. The third complex is normal. The R is high and measures 0.06 second. This is followed by three abnormal complexes like the first two and then again a normal complex. The second normal complex is followed by three abnormal complexes after which comes the third normal complex. The fourth and fifth normal complexes are also preceded by three abnormal complexes. The P in this lead can be measured fairly accurately. The ventricular rate is 140.3 per minute. The P-R and R-R intervals are as follows:

P-R	1.	.125	R-R	1.	.430
	2.	.120		2.	.435
	3.	.140		3.	.420
	4.	.140		4.	.425
	5.	.120		5.	.435

P-R	6.	.130	R-R	6.	.430
	7.	.130		7.	.415
	8.	.120		8.	.425
	9.	.115		9.	.430
	10.	.125		10.	.440
	11.	.125		11.	.410
	12.	?		12.	.440
	13.	.120		13.	.425
	14.	.120		14.	.435
	15.	.120		15.	.410
	16.	.125		16.	.435
	17.	.130		17.	.435
	18.	.125		18.	.430
	19.	.125		19.	.415
	20.	?		20.	.420

The QRST of the abnormal complexes measures 0.320 second, and that of the normal complexes 0.285 second, a difference of 0.035 second. The third lead shows the first complex with a deep S, notched at its apex, the entire QRS widened and the T oppositely directed, coming off directly from the upstroke of the S. The next R is low but of normal configuration and narrow so that the QRS measures 0.05 second. Its T is low and entirely different from the previous T. This is followed by three complexes like the first one, after which a complex like the second or the normal occurs. This second normal is followed by three abnormal complexes and then a third normal complex. The ventricular rate is 135.6 per minute. The P-R and R-R intervals measure as follows:

P-R	1.	.140	R-R	1.	.440
	2.	.145		2.	.425
	3.	.135		3.	.430
	4.	.135		4.	.440
	5.	.140		5.	.440
	6.	.140		6.	.430
	7.	.130		7.	.435
	8.	.145		8.	.430
	9.	.145		9.	.440
	10.	.145		10.	.430
	11.	.130		11.	.445
	12.	.140			

How is this to be interpreted? The first three complexes of lead one and all those similar in that lead, the first two complexes in lead two and all those similar in lead two, and the first complex in lead three and all those similar in lead three indicate that there is a disturbance of conduction in the right branch of the bundle of His. These complexes are constantly present and are the only ones present in every electrocardiogram taken on this patient after January 8, 1930. These complexes are widened much beyond 0.1 second, they are diphasic in leads one and three and also in lead two in many of the subsequent records. The most important characteristics of bundle-branch block are the widening of the complex beyond 0.1 second and an oppositely directed T, constituting a diphasic complex. The height is influenced by

many factors.[35] The complex would have been higher and broader but for the fact that the heart of the patient was small. The left ventricle was not enlarged. This is extremely important for the size and width of the complexes (Wenckebach & Winterberg[37]). The x-ray examination of the heart and the clinical findings all reveal the heart of normal size and shape. It is known that in typical bundle-branch block produced on the dog that the complexes may be lower than those before the cut was made.[35] How are we to regard complexes four, eight, eleven and fourteen of lead one, complexes three, seven, eleven, fifteen and nineteen of lead two and complexes two, six and ten of lead three? Leads two and three show that all complexes, the abnormal and the normal are preceded by a P-wave. In lead one this also is so, but it is not sufficiently clear. The assumption is that all the complexes are therefor of supraventricular origin. I believe that these complexes are normal in appearance because there is the algebraic summation of a dextrocardiogram and a levocardiogram, for I believe that the impulse traveled down from the A-V node and without being blocked this time descended both branches and activated both ventricles simultaneously. The abnormal complex is that of a levocardiogram due to temporary block of the right branch of the bundle of His. This block lasts either 1.71 to 1.72 seconds or occasionally 1.255 seconds requiring almost all the time with two exceptions, the former figures for recovery.

There is only one possibility which suggests itself in view of the work of Wilson and Herrmann[36] but if directed against my interpretation can easily be answered. The possibility may exist that an extrasystole may occur just below the point of blockage in the right branch, come at the exact time to complete the activation of the right ventricle at exactly the same time as the activation of the left ventricle from the supraventricular impulse and produce the normal cardiogram. If the figures of the R-R interval are looked into, it will be seen that the R-R interval preceding the normal complex is always longer and by a good deal over that of the R-R interval following the normal complex. If this were an extrasystole, the opposite should be true. As a matter of fact, in the curves shown by Wilson and Herrmann[36] measurement shows that the pause is almost every time longer after the extrasystole is provoked than the period before and never shorter. A point might be raised that the R-R interval is shorter after the normal complex than before the normal complex because the R-R interval before includes the widened QRS of the complex that precedes the normal one. The following figures chosen at random but illustrating what occurs in all of them is shown. The interval R-R before complex seven of lead two is 0.430 second. The following R-R is 0.415 second. Now if 0.06 second were added it would make it 0.421 second the 0.06 second being the difference between the normal and the ab-

normal complex. Nor is this changed by the P-R interval. The P-R interval of the normal complex is 0.130 second and of the next complex 0.120 second, so that in this case 0.01 second might also be added. All in all the R-R interval after the normal beat is still shorter than the R-R interval before the normal beat. As a matter of fact, every measurement throughout the record shows this same state of affairs, that is, a much shorter time of the R-R interval after the normal complex than the previous R-R interval. Not only that but the figure of the R-R interval after each normal complex is below the general average. I believe that it is due to the influence of the extracardiac nerves and it may be the factor which caused conduction to improve in the right branch of the bundle of His and transmit the supraventricular impulse.

The second point against the conception of an extrasystole coming at such a time so as to form a normal complex is the P-R interval. In no place where it can be measured accurately does the normal R come before the average time. There is no shortening of the P-R interval indicative of an extrasystole arising late in diastole.

If these are extrasystoles arising in the right branch in the bundle of His below the block, we might expect, though not always, a definite coupling with the previous beat. In lead one the R-R interval **3** is 0.440 second, R-R **7** is 0.435 second, R-R **10** is 0.425 second, R-R **13** 0.435 second. The difference is 0.15 second. In lead two the difference is 0.10 second and in lead three no variation. If the coupling is constant, differences as much as 0.15 would have produced enormous differences in the summation in the dextrocardiogram and levocardiogram. Wilson and Herrmann[36] showed that retardation of as little as 0.005 second of either the levocardiogram or the dextrocardiogram produced very definite differences in the complexes. There is no need to go into this any further. All the normal complexes are similar in each of the leads.

Another fact is that these normal complexes are too regular and constant in their occurrence and configuration.

I do not believe one can seriously doubt that they are normally conducted impulses from a supraventricular origin in both branches of the bundle of His. Fig. 2 indicates what I believe has taken place. We thus have a case of three-to-one and four-to-one right bundle-branch block comparable to that of auriculo-ventricular block of the same nature. The closest record to mine is one reported by Hewlett.[10] He shows, in his diagrams a levocardiogram and then a normal complex, or two levocardiograms and then a normal complex. But in his case the cycle before the extrasystole is short and that after the extrasystole longer than any R-R interval, making a compensatory pause. Furthermore there is a shortened P-R interval. There is no doubt of

his case being extrasystolic in nature, but my record shows nothing like this. In Stenström's last case he showed a five-to-three block in one part of his record.[31]

Such a diagnosis was suspected when the record was first seen and with this in mind the patient was recalled that evening and another tracing taken (Fig. 3). As can be seen the complexes were all of those of right bundle-branch block. The ventricular rate in leads one and two was 117.6 per minute and toward the end of a lead three 127.6 per minute. It would be natural to expect that with the lower rate conduction would improve but it did not. An attempt was made to alter the chronotropic, dromotropic and bathmotropic influences. This

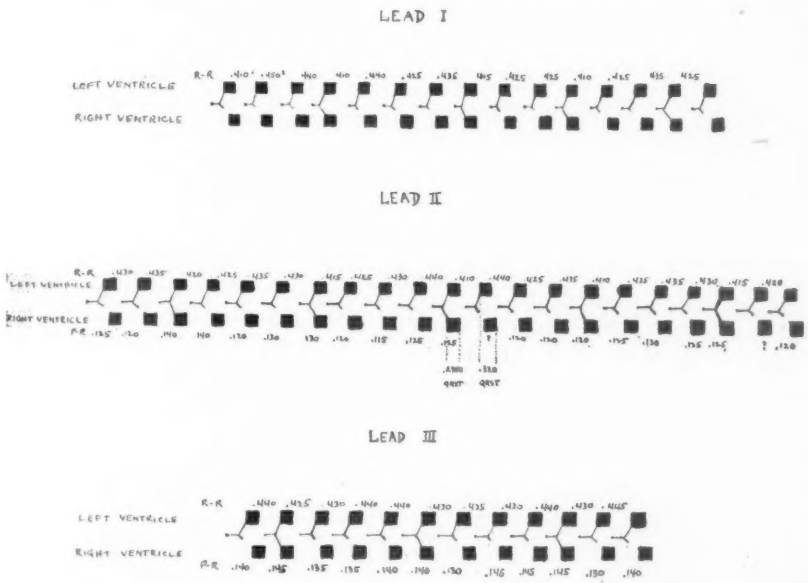


Fig. 2.—The diagram is not meant to show P-R conduction. It only illustrates the conduction from the auriculo-ventricular node down the common branch and into the right and left branches. The P-R intervals mentioned numerically under each lead are for the purpose of quick orientation.

was done for the entire next month but without succeeding in obtaining another record like that of Fig. 1. That night she was exercised, vagal pressure aplied and gynergen given. Conduction in the right branch of the bundle of His could not be improved (Fig. 4). Because the response was not as definite and perhaps not carried out under the best possible conditions, comment will not be made on this record.

On January 17, 1930, the electrocardiogram showed a typical right bundle-branch block with a ventricular rate of 101.7 per minute (Fig. 5). On that day 0.5 c.c. of adrenalin was given subcutaneously. The record shows that the rate went up to 122.4 per minute and later returned to 90.7 per minute with a short P-R interval and higher QRS

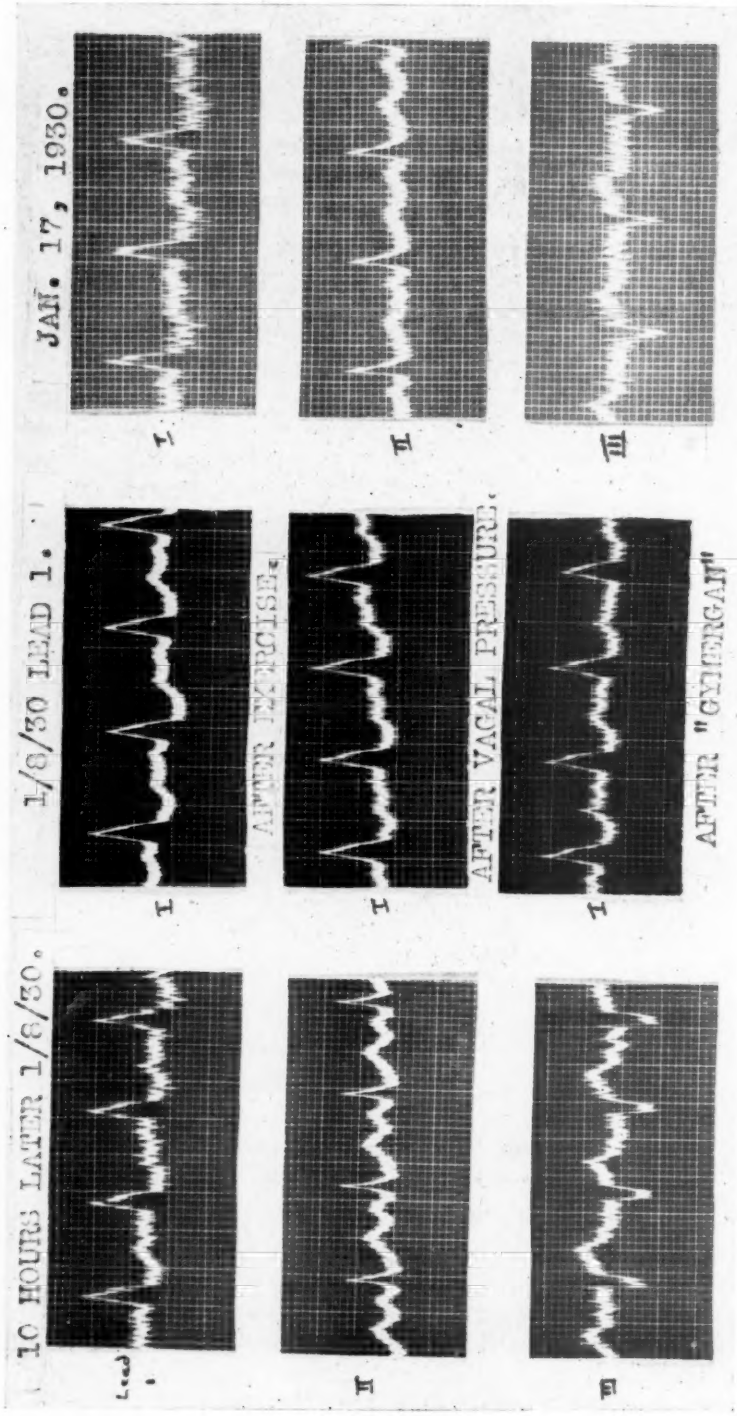


Fig. 3.

Fig. 4.

Fig. 5.

complexes (Fig. 6). On January 21, 1930, another electrocardiogram was taken which showed a ventricular rate of 101.4 per minute. Its other characteristics were as before. On the same day after Fig. 7 was taken, various tests were tried, the responses to which are shown in Fig. 8. These responses are characteristic of all the reactions which were obtained in the hospital. On vagal pressure there was practically no slowing. After exercise the rate went up to 171.4 per minute. This represents only part of the record. In view of the fact that the rate of 140.3 per minute seemed best for the production of a rhythm like Fig. 1, continuous strips after exercise were taken repeatedly so that when the rate returned to the above figures the rhythm such as that of Fig. 1 might return. However it never did.

On January 22, 1930, 0.5 c.c. of gynergen was given subcutaneously with the usual typical result. The rate before injection was 117.6 per minute and rose to 136.3 per minute (Fig. 9). Fig. 10, taken on January 28, 1930, shows a typical right bundle-branch block. Fig. 11 was taken January 30, 1930. Notice that the ventricular rate is 142.6 per minute, almost the same rate as of Fig. 1, and yet an extremely long strip did not reveal any improvement in conduction.

All these records are reproduced for the purpose of showing her subsequent course and the inability to improve conduction by all the methods tried. It is not our purpose in this paper to discuss the pharmacological aspects of her response to the various drugs.

If now a normal complex is taken from each of the three leads, it can be seen that there is no left ventricular preponderance, confirming the clinical and roentgen findings. This accounts for the QRS complexes of the blocked impulses being not particularly pronounced.

The first efforts at direct investigation of disturbances in the conduction in the branches of the bundle of His arose after the work of Eppinger and Rothberger in 1910.[6] They injected silver nitrate into the muscle of the heart with the purpose of ascertaining what the effect of involving of a large area of the heart would be on the electrocardiogram. They were struck by the fact that in certain instances a marked disproportion existed between the amount of muscle involved and the electrocardiograph findings. If a small area were involved near the branches of the junctional system, a marked change was noted. They[7] followed this with experiments directed to cutting either branch of the bundle of His, and obtained curves characteristic of the lesion. Eppinger and Störk[8] then reported five cases in the human being in which two show post-mortem verification. The work was continued by Rothberger and Winterberg.[26] From then on until the present time the Viennese school have been intensely interested in conduction disturbances in the bundle of His and its branches.

Lewis[16] also obtained experimental corroboration and showed that the normal electrocardiogram was the algebraic summation of a dex-

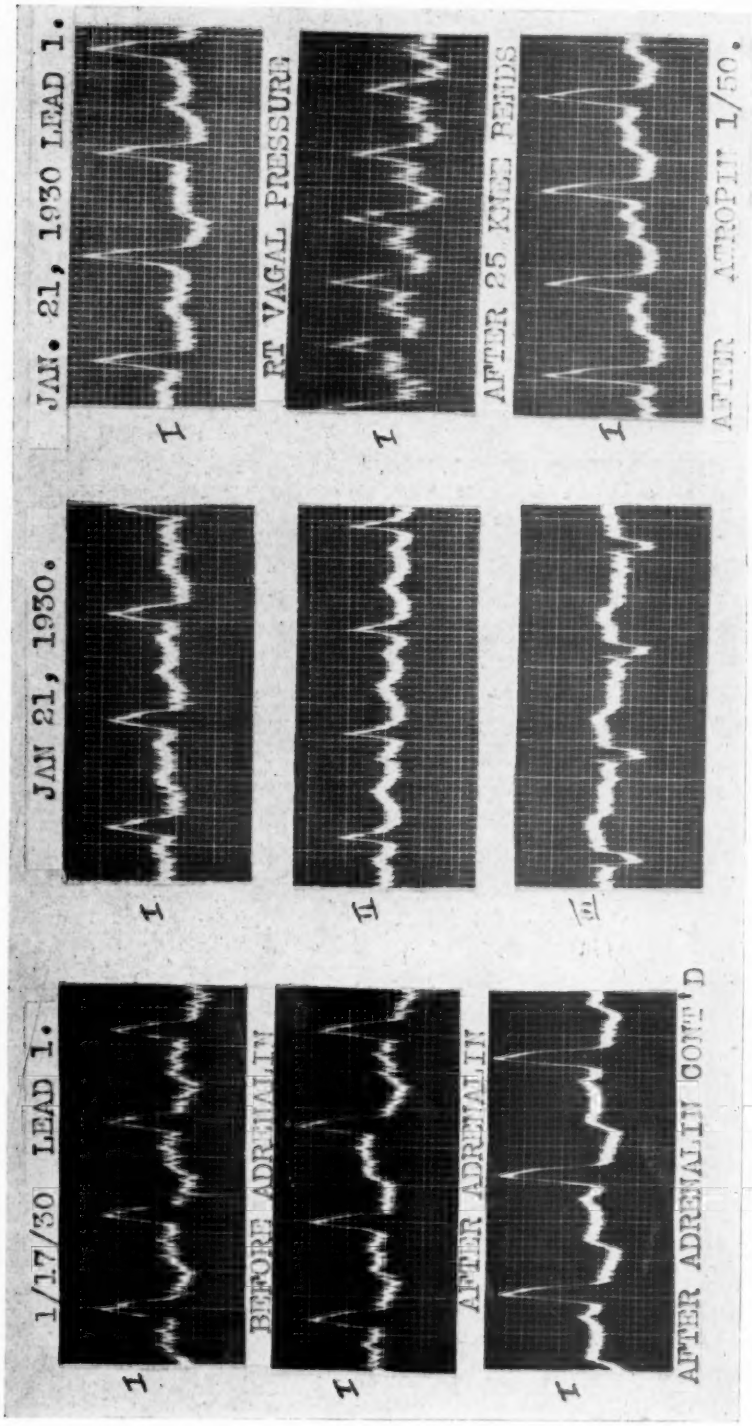


Fig. 6.

Fig. 7.

Fig. 8.

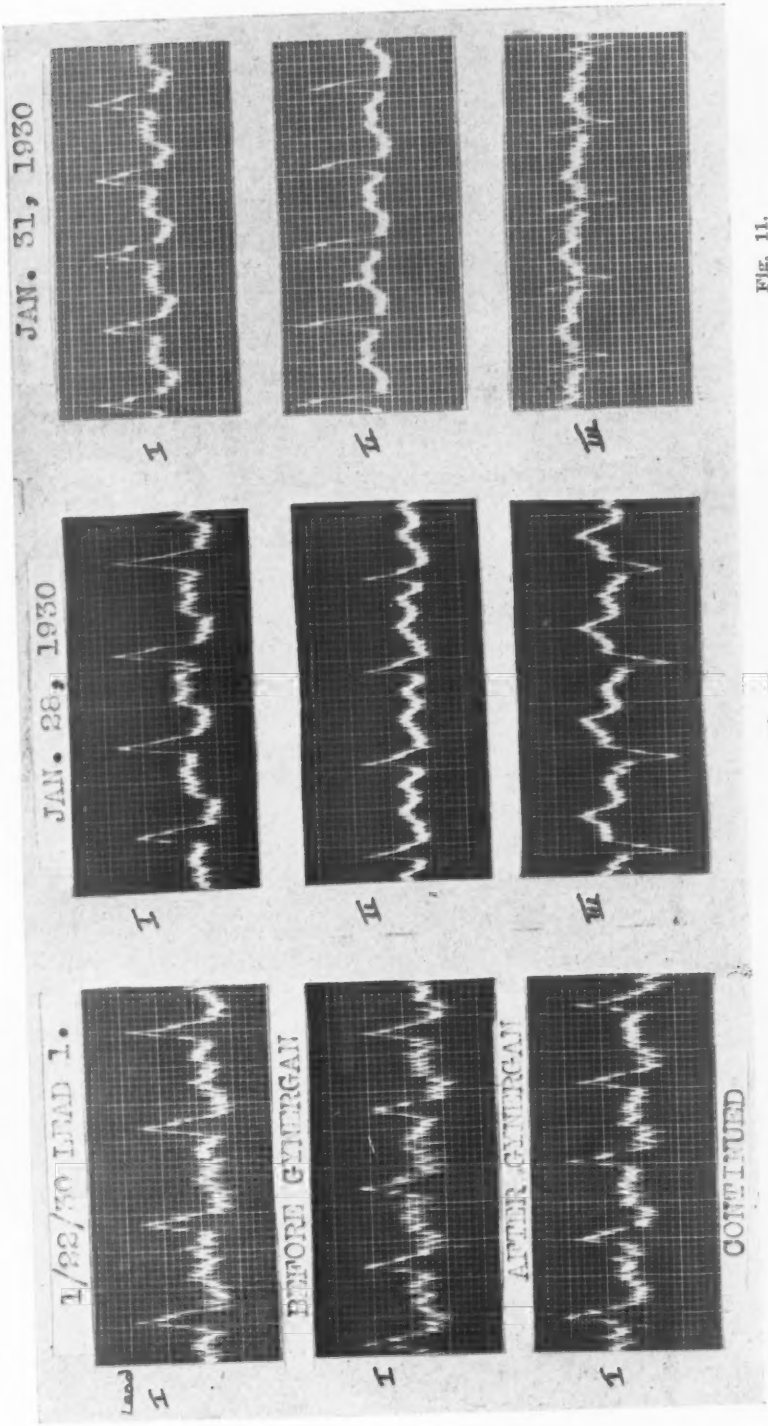


Fig. 9.

Fig. 10.

Fig. 11.

trocardiogram and a levocardiogram. At this time various papers of Christian,[2] Cohn,[4] Mathewson,[18] Fridericia and Möller,[9] Robinson[22, 23] and Wilson[34] aroused the keenest interest in the possibility of incomplete block of either branch of the bundle of His. This led to a most brilliant piece of experimental work on this phase of the question by Wilson and Herrmann.[36] Then Stenström[29] reported his first paper on incomplete bundle-branch block.

From then on tremendous clinical corroboration was obtained and the condition of bundle-branch block invited closer study, both experimental and clinical. It was then learned that blocks may occur in either branch which resemble auriculo-ventricular block. The work of Scherf[26] and Stenström[30] stands out.

Of the various classifications of A-V block, such as that of Wenckebach and Winterberg,[37] and Lewis, it is apparent that two broad divisions may occur. They are the complete block and the incomplete block. The incomplete auriculo-ventricular blocks are further subdivided into those without dropped beats and those with dropped beats. The latter, called partial heart-block, is again divided into those in which the dropped beats occur with a gradual increase of conduction time until a point is reached whereby the stimulus is completely blocked. The other type of dropped beats occurs without any warning so to speak; the conduction time is normal but a stimulus is suddenly blocked. This is the Hay's type. According to Mobitz, the latter is called type II heart-block.

This classification is necessary for the understanding of conduction disturbances in either branch of the bundle of His. I believe with the report of my case, which is the first of its kind on record, not only clinically but also experimentally, to have filled in the last gap in the chain which completes the resemblance to auriculo-ventricular block.

Blockage in the bundle can therefore be compared kind for kind and type for type with auriculo-ventricular block with certain differences which I will explain further on. These differences are due to the anatomical arrangement of the bundles and the method of supply to the ventricles of the impulse. In order to clarify the above classification, I will use incomplete bundle-branch block in the sense of simple prolongation of conduction as compared to a prolonged P-R interval, partial bundle-branch block for the dropped beats as compared to that in auriculo-ventricular partial heart-block with dropped beats. The partial bundle-branch block is further subdivided into those cases with gradual increase of conduction time and finally dropped beats, the so-called "Wenckebach Periods" and the dropped beats without prolongation of conduction time. Then there is the complete bundle-branch block.

CLASSIFICATION OF BUNDLE-BRANCH BLOCK

I. Incomplete bundle-branch block.
II. Partial bundle-branch block.
  A. With formation of "Wenckebach Periods."
  B. Without prolongation of conduction time (Type II, Mobitz).
III. Complete bundle-branch block.

Incomplete Bundle-Branch Block.

This type of bundle-branch block is compared to that of simple prolongation of the P-R interval in auriculo-ventricular block. It is characterized in its analogy by the increase in the QRS time. Some very important points should be mentioned at this point. When the impulse reaches the bifurcation of the bundle of His, it travels down each branch producing a dextrocardiogram and a levocardiogram which algebraically summated produced the normal electrocardiogram. If the passage is delayed in the slightest degree, the impress of the intact cardiogram dominates the picture. The delay in either branch of the impulse by as little as 0.005 second is sufficient to cause the electrocardiogram to change and begin to assume characteristics of the intact ventriculogram (Wilson and Herrmann[36]). In other words the normal electrocardiogram is divided into its two components more and more as the impulse is retarded in the diseased or the cut branch of the bundle of His. Now in the dog, as worked out by Lewis and Rothschild, by estimating the delay of the wave of negativity, they found a difference between the intact and severed ventricle of about 0.03 second. These figures corresponded to those of Wilson and Herrmann, who estimated in another way, that is, by the refractory period that the delay was the same. There is no direct way of estimating this in the human being. However, because of the larger heart and the longer system of branches of the bundle of His in the human being, we can estimate indirectly by the speed of conduction and the difference in length that the delay of the impulse in the ventricle with the severed bundle should reach between 0.035 and 0.05 second. This at the best is only a crude way and the results are not accurate.

If the conduction in either branch in the human being is delayed more than from 0.035 to 0.05 second, the picture produced on the electrocardiogram is that of complete bundle-branch block. The duration of the block, however, in the branch may be anywhere from 0.05 second to one second; for if the conduction time can be prolonged so greatly in auriculo-ventricular block (cases have been reported of that length) there seems no reason why such delay should not occur in either branch of the bundle of His. The anatomical distribution of the fibers and the heart itself causes this difference, however. If the impulse is delayed in either branch, the intact branch activates its

own ventricle and the other ventricle is activated through the septum indirectly. After from 0.035 to 0.05 second the injured ventricle has been completely though indirectly activated. Still its own bundle may be "passable"; that is, the impulses may be conducted through it, but by the time it could do it the impulse has passed from the intact ventricle through the septum and the injured ventricle already activated. The impulse, so to speak, is frustrated in the injured branch. Two important facts follow from this. First innumerable cases which are called complete bundle-branch block are in reality incomplete bundle-branch block. And secondly, there is a matter of only 0.035-0.05 second for the play of the various types of incomplete bundle-branch block as noted in the classification above to make itself manifest. This latter fact is what has made it so hard to find clinical examples of the various types of block to correspond to those of the A-V block.

With this in mind we must go back to the earliest descriptions of bundle-branch block. Lewis noted in auricular extrasystoles that very often there was an aberrancy of the ventricular complex. In addition it was later noted that in various types of auricular tachycardia, mostly in auricular flutter that the ventricular complex assumed an aberrant form. The work of Nils Stenström[29] should be mentioned in this place. In one of his cases with the beginning of the auricular tachycardia, the cardiogram was normal, but as the tachycardia continued the cardiogram assumed aberrancy. These clinical cases have been reported repeatedly. The aberrancy consisted in the ventricular complex assuming a form like that of a dextrocardiogram or a levocardiogram, which meant that in these cases there was either a delay or a complete block in either branch in the bundle of His. In auricular extrasystoles, its early occurrence or a slow recovery time in either branch, loaded this particular branch and conduction was blocked or delayed. The same applies to those cases of auricular flutter and tachycardia. It is difficult to state whether the impulse is delayed beyond the given time or whether the block is complete because in the electrocardiogram there are no definite criteria if the picture is that of complete bundle-branch block. This also applies to those cases in which there was bundle-branch block at one time (Willius and Keith[33] and Kapff[12]) and at a future time this had disappeared. We know that complete A-V block can recede. However logical it may appear to assume that if the block appears and disappears in very short intervals, we are dealing with incomplete heart-block, there is not the finality such as we would find in simple prolongation of the P-R interval.

But many cases have been reported of transitional forms between a normal cardiogram and a dextrocardiogram or levocardiogram. As is often seen in hospital records; there is widening but the T-wave does not make a diphasic curve. Or records are seen which look inter-

mediary, that is, there is not enough widening of the QRS with an inverted T, and subsequently, when a record is taken on the same patient, we now find a picture of complete bundle-branch block. Cases have been reported in which the vagus played an important rôle in delaying conduction in one of the branches of the bundle of His. In the course of vagal pressure the electrocardiogram took the form of bundle-branch block. Often intermediary pictures were obtained, only to recede quickly, which certainly indicated delay of conduction in one of the branches. In the beautiful pictures of Lutembacher[16, 17] where schematic representation of delay of one cardiogram in certain time increments produces variations in the cardiogram, the picture we often see in our own records can be found where the delay of conduction lies within 0.03 second.

Another point to be mentioned is that the so-called arborization block is being interpreted because of experimental corroboration[35, 36] as incomplete bundle-branch block.

Incomplete bundle-branch block is not at all uncommon and many cases of so-called complete bundle-branch block are only higher forms of incomplete bundle-branch block.

Partial Bundle-Branch Block.

If in conduction disturbances three factors and even a fourth are important, namely, the strength of the stimulus, the conductivity, the irritability and finally contractility, the classification is justified. In the division of partial heart-block or where there are dropped beats of two types, namely, the one with gradual increase of conduction time and then the drop of a beat and the other of dropped beats without prolongation of conduction time, it is possible that the factors above mentioned are of the utmost importance.

In comparing a growing P-R interval (Wenckebach Periods) until the drop of a beat with that of partial bundle-branch block of this type, we must remember that the QRS is comparable to the P-R. As conduction is delayed more and more in one branch, the QRS grows wider and wider because of the contralateral activation, until the full picture of a complete bundle-branch block is obtained. It is therefore necessary that in partial bundle-branch block of type I, that the QRS grow wider and wider until a complete dextrocardiogram or levocardiogram is obtained and then suddenly the next cardiogram is normal. This occurs because as the last impulse to the bundle was blocked, the bundle rested and the next impulse from the A-V node spread normally producing a normal cardiogram. Records of clinical cases have been reported (Christian[2] and Wilson[34]).

Cases of two-to-one partial bundle-branch block have been reported. The electrocardiogram shows a normal cardiogram followed by a dextrocardiogram or a levocardiogram with similar R-R periods. Three cases are mentioned in the textbook of Wenckebach and Winterberg,[37]

one by Stenström[31] and one by Leinbach and White.[13] In what light are we to interpret these? Are they of type I or type II? If there were a three-to-two block, it would be easy, for in the two curves which transmitted the impulse the growth of the QRS interval would show whether the so-called periods were being formed. But in two-to-one block this is not possible. So these cases might be of either type. In the case of Stenström[31] he shows records (see plates 18 and 19) in which there are two normal and one abnormal cardiogram and calls this a three-to-one block. This is a misnomer and he admits in his text that it may be so. It should be called two-to-three block. Let us compare this with A-V block. If it were of the type in which we have three auricular beats which produce two consecutive responses in the ventricle and a third beat is blocked, we should be dealing with a three-to-two block, that is one dropped beat. If the two responses of the ventricle show increasing P-R intervals before the dropped beat it is of type I partial heart-block. If the P-R is constant in the two transmitted beats and the third is dropped it is of type II. In Stenström's record only the third beat was dropped but two consecutive beats showed no increase in the QRS and the third beat which was blocked came without warning, so to speak. We are therefore dealing in just this isolated part of his record with a three-to-two partial bundle-branch block of type II.

This brings us to our record. It shows both three-to-one and four-to-one partial bundle-branch block. Two or three impulses are blocked in the right branch but a third or fourth is conducted through normally. The block shows intermittence. Immediately after the normal beat, without warning, the next two or three impulses in the right branch are blocked. If we compare this to A-V conduction, that is, the P-R interval is normal and then suddenly two or three auricular beats are completely blocked it would not belong to type I, for then there should be a growing QRS interval beginning with a normal or almost a normal QRS immediately after the conducted impulse. In our record this is not so. The QRS immediately after conduction is at once raised to that of all the others which are blocked. This case, therefore, belongs to type II of partial bundle-branch block and fulfills requirements mentioned by Wenckebach and Winterberg[37] at the bottom of page 360: "Intraventricular block of type II can on the other hand be confirmed when, without dropping of ventricular systoles, one normal electrocardiogram follows two or more similar atypical complexes. No known clinical example has come to our notice."

If the factor of irritability in this case is used, it might be possible to give some explanation of what has happened in this case. It would seem that the irritability was of a very low degree and when the impulse was conducted through the right branch, the threshold was raised

high enough by the previous rest to enable the impulse to pass. With the conduction, irritability was exhausted and a rest period of usually 1.712 seconds or occasionally 1.25 seconds was sufficient for the threshold to reach the height necessary for conduction again to take place. If in addition in this case the strength of the stimulus were low it would form an additional factor for the disturbance in conduction. This patient had myocardial involvement.

In an attempt to bring out conditions which might influence any of the factors of conduction the patient was submitted to various tests. However, they were all unsuccessful in improving conduction.

SUMMARY

1. A case of three-to-one and four-to-one partial bundle-branch block of type II is reported which we believe is the first of its kind in the literature.

2. Some phases of incomplete and partial bundle-branch block are briefly discussed.

REFERENCES

1. Barnes, Arlie R., and Yater, Wallace M.: Paroxysmal Tachycardia and Alternating Incomplete Right and Left Bundle-Branch Block with Fibrosis of Myocardium. Medical Clinics of North Amer., **12**: 1603, 1929.
2. Christian, Henry A.: Transient Auriculo-ventricular Dissociation with Varying Ventricular Complexes Caused by Digitalis. Arch. Int. Med., **16**: 341, 1915.
3. Coelho, Eduardo: Isolated Block of Branches and Endings of Bundle of His. Lisboa Med., **5**: 638, 1928.
4. Cohn, Alfred E.: A Case of Transient Complete Auriculo-ventricular Dissociation, Showing Constantly Varying Ventricular Complexes. Heart, **5**: 5, 1913-14.
5. Danielopolu, D., and Danulesco, V.: Disturbances of Conduction in the Branches of the Auriculo-ventricular Bundle Produced in the Normal Man by Vagal Excitation. Arch. d. mal. du coeur, **15**: 361, 1922.
6. Eppinger, H., and Rothberger, C. J.: Analysis of Electrocardiogram. Wien. klin. Wchnschr., **22**: 1091, 1909.
7. Eppinger, H., and Rothberger, C. J.: Consequences of Transecting the Tawaran Branch of the Conduction System. Ztschr. f. klin. Med., **70**: 1, 1910.
8. Eppinger, H., and Störk, O.: Clinical Significance of Electrocardiogram. Ztschr. f. klin. Med., **71**: 157, 1910.
9. Fridericia, L. S., and Möller, Paul: A Case of Localized Myocarditis of the Ventricular Septum with Peculiarities in the Electrocardiogram. Deutsch. Arch. f. klin. Med., **126**: 246, 1918.
10. Hewlett, Albion Walter: A Case Showing Bundle-Branch Block with Extrasystoles Originating in the Ventricular Septum. Heart, **9**: 1, 1921-1922.
11. Kahn, R. H.: Electrocardiographic Studies. Pflügers Arch., **140**: 627, 1911.
12. Kapff, W. V.: A Case of "Passable" Bundle-Branch Block. Klin. Wchnschr., **7**: 357, 1928.
13. Leinbach, R. F., and White, Paul D.: Two-to-One Right Bundle-Branch Block. Am. Heart J., **3**: 422, 1928.
14. Lewis, Thomas: Paroxysmal Tachycardia, the Result of Ectopic Impulse Formation. Heart, **1**: 262, 1909-10.
15. Lewis, Thomas: The Mechanism and Graphic Registration of the Heart Beat, 3rd Edition, Shaw and Sons Ltd., London, 1925.
16. Lutembacher, R.: Disturbances of Conduction of the Right Branch of the Bundle of His and Paroxysmal Auricular Tachycardia. Arch. d. mal. du coeur, **16**: 120, 1923.
17. Lutembacher, R.: The "Complimentary" Ventricular Complexes. Arch. d. mal. du coeur, **16**: 241, 1923.

18. Mathewson, George D.: Lesions of the Branches of the Auriculo-ventricular Bundle. Heart, **4**: 385, 1912-13.
19. Meyer, Fritz: An Apparatus for Demonstrating the Construction of Interference Curves and Its Use for Explaining the Ventricular Electrocardiogram. Pflügers Arch., **216**: 765, 1927.
20. Oppenheimer, B. S., and Williams, H. B.: Prolonged Complete Heart-Block without Lesion of the Bundle of His and with Frequent Changes in the Idioventricular Electrical Complexes. Proc. Soc. Exp. Biology and Med., **10**: 86, 1912-13.
21. Resnick, William H.: Observations of the Effect of Anoxemia on the Heart. II. Intraventricular Conduction. J. Clin. Invest., **2**: 117, 1925.
22. Robinson, G. Canby: The Relation of Changes in the Form of the Ventricular Complex of the Electrocardiogram to Functional Changes in the Heart. Arch. Int. Med., **18**: 830, 1916.
23. Robinson, G. Canby: The Significance of Abnormalities in the Form of the Electrocardiogram. Arch. Int. Med., **24**: 422, 1919.
24. Rothberger, C. J., and Winterberg, H.: The Diagnosis of Block of Conduction in One of the Branches of the Tawaran Bundle. Zentralbl. f. Herz und Gefässkrank., **5**: 206, 1913.
25. Scherf, D.: Conduction Disturbances in Bundle. Klin. Wchnschr., **4**: 2207, 1925.
26. Scherf, D.: Experimental and Clinical Investigations on Intraventricular Conduction Disturbances. Wien. Arch. f. inn. Med., **14**: 433, 1927.
27. Scherf, D.: Conduction Disturbances in Heart. Wiener klin. Wchnschr., **41**: 375, 1928.
28. Samet, B.: Intraventricular Conduction Disturbances. Wien. Arch. f. inn. Med., **14**: 15, 1927.
29. Stenström, Nils: Contribution to the Knowledge of Incomplete Bundle-Branch Block in Man. Acta Med. Scand., **57**: 385, 1922-23.
30. Stenström, Nils: An Experimental and Clinical Study of Incomplete Bundle-Branch Block. Acta Med. Scand., **60**: 552, 1924.
31. Stenström, Nils: Further Experience in Incomplete Bundle-Branch Block. Acta Med. Scand., **67**: 353, 1927.
32. Weisser, Egon: The Interference of Two Excitation Waves in the Human Ventricle of the Heart. Zentralbl. f. Herz u. Gefässkrank., **11**: 197, 1919.
33. Willius, Frederick A., and Keith, Norman M.: Intermittent Incomplete Bundle-Branch Block. Am. Heart J., **2**: 255, 1927.
34. Wilson, Frank N.: A Case in Which the Vagus Influenced the Form of the Ventricular Complex of the Electrocardiogram. Arch. Int. Med., **16**: 1008, 1915.
35. Wilson, Frank N., and Herrmann, George R.: Bundle-Branch Block and Arborization Block. Arch. Int. Med., **26**: 153, 1920.
36. Wilson, Frank N., and Herrmann, George R.: An Experimental Study of Incomplete Bundle-Branch Block and of the Refractory Period of the Heart of a Dog. Heart, **8**: 229, 1921.
37. Wenckebach, K. F., and Winterberg, Heh.: Irregularities of the Heart. Wilhelm Engelmann, Leipzig, 1927.
38. Winterberg, Heinrich: Disturbances of Stimulus Conduction in the Human Heart and the Attacks in Adams-Stokes Symptom Complex. Ztschr. f. d. Gesamte. Exp. Med. **8**: 131, 1919.

THE LOCAL AND SYSTEMIC EFFECTS OF ARTERIO-VENOUS FISTULA ON THE CIRCULATION IN MAN*

LAURENCE B. ELLIS, M.D., AND SOMA WEISS, M.D.
BOSTON, MASS.

INTRODUCTION

ONLY within recent years has it been realized what profound systemic effects acquired arterio-venous fistulae may exert on the human circulation. During the past decade, however, experimental and clinical studies by Reid,[1] Holman,[2] Matas,[3] Lewis and Drury[4] and others have demonstrated that such abnormal communications between the arterial and venous portions of the vascular circuit may so severely damage the heart and peripheral circulation that chronic invalidism and even death may result. It has, therefore, generally been conceded that operative interference for the relief of such fistulae is desirable not only to alleviate the local condition but also to avoid or remedy general circulatory failure.

Although much information has been gained as to the functional pathology of the circulation in this condition, the exact mechanism of many of the circulatory phenomena commonly observed is disputed. In particular, there has been little direct experimental study in man of changes in the cardiac output, in venous pressure, and in the circulation in tissues adjacent to the fistula. The purpose of this investigation was to repeat certain of the observations previously made and to obtain further knowledge of the circulatory changes in arterio-venous aneurism by the application of recently introduced technical procedures. In the cases here reported it was possible to study the effect on the circulation of suddenly eliminating the aneurism by manual compression. Moreover, both patients were carefully observed following operative excision of the fistulae. These post-operative observations throw objective light on the value of surgical elimination of the arterio-venous fistula. This unusual opportunity for observing patients in whom the whole circulation may be profoundly altered, either acutely by manual compression, or permanently by operation, makes the study of arterio-venous aneurism of especial importance. Such study may throw light on the physiology of the circulation in general, and, in particular, on types of cardiovascular disease presenting analagous circulatory phenomena.

The two cases of arterio-venous aneurism studied were similar in being of traumatic origin and of short duration. One patient had a fistula sufficiently extensive to produce the classical effects on the general cir-

*From the Thorndike Memorial Laboratory, Boston City Hospital and the Department of Medicine, Harvard Medical School.

culation; the second showed no or slight systemic disturbance, but presented important local vascular changes. The clinical and experimental records of these patients will be presented separately.

Case I

(a) *Clinical History and Physical Findings.*—The patient (J. L.) was a butcher of 23 years whose family and past history was negative. Twenty-nine days before entry, on September 14, 1929, while cutting meat, he accidentally stabbed himself over the right femoral vessels. He lost a considerable amount of blood at the time and fainted. He was later transfused and remained in bed. He was weak from loss of blood; conscious of a pulsation in his right groin; and had considerable pain in his right leg and foot. This pain was particularly marked on holding the foot in a dependent position. There were slight cardiac symptoms. Examination revealed a well-developed but somewhat pale young man. The lungs showed no abnormality. There was marked pulsation in the carotid, brachial and radial arteries. The left border of the heart was just outside the nipple line. The sounds were loud and there was a systolic murmur at the apex which was also heard over the carotid and subclavian arteries. Both radial pulses were collapsing in quality and there was marked capillary pulsation. Over the right groin, below Poupart's ligament, there was an elevated pulsating area over which there was a continuous thrill and murmur with systolic accentuation. The thrill and murmurs also extended over a considerable area beyond the tumor, particularly downward, nearly to the knee. The skin surrounding the tumor was definitely warmer than on the unaffected side.

The patient remained in bed for three months in order to establish adequate collateral circulation. During this time his anemia diminished greatly. He was constantly aware of a forceful heart action. The pain in his right leg became less, but otherwise he showed no subjective or objective change. On December 5, 1929, Dr. William Morrison performed a quadruple ligation of the right femoral artery and vein, under ether anesthesia, and removed a saccular aneurism about four centimeters in diameter with an opening between the artery and vein about three millimeters in diameter. Following this, convalescence was uneventful except for slight infection in his wound. There was no evidence of deficient circulation of the right leg. The palpitation disappeared. Examination three weeks after operation showed that he was less pale than at the first examination. There was no capillary pulsation, nor visible pulsation of the peripheral arteries. A slight pistol shot was heard over the brachial and left femoral arteries. The left border of the heart was percussed the same distance from the sternum as formerly. The sounds were normal and no murmurs were heard over the heart or vessels of the neck. Over the right Scarpa's triangle there was a scar about five inches long in which were two small areas of infection. There was no tumor, thrill or murmur. The remainder of his convalescence was uneventful. Six weeks after operation he sat up and was discharged well three weeks later.

(b) *Circulatory Measurements.*—Certain of the circulatory measurements which were performed are tabulated in Table I. Where possible these measurements were repeated while the aneurism was completely occluded by manual compression over the fistula, and again following the operation. The figures given in many instances represent the average of repeated observations.

Pulse Rate.—Upon compression of the aneurism the heart rate per minute dropped from 80, which is slightly above the average normal for a man under basal conditions, to 60. This phenomenon of an immediate slowing of the cardiac rate upon occluding the fistula is generally known in this country as the Branham Bradycardia Phenomenon. The importance of this finding has been stressed not only

as a valuable diagnostic point in this condition, in particular as opposed to purely arterial aneurisms, but as an aid in evaluating the extent of existing systemic involvement.

Arterial Blood Pressure.—The measurement of the arterial blood pressure was accomplished both by a mercury sphygmomanometer and by a Tycos recording sphygmomanometer. The chief point of significance is the low diastolic pressure which was found. Upon occlusion of the aneurism there was a slight elevation of the systolic and a definite rise in the diastolic pressure, with a consequent increase in the mean pressure. Following operation, the average systolic pressure level was slightly lower than it had been previously, whereas the diastolic pressure had returned to a normal range.

TABLE I

CIRCULATORY MEASUREMENTS IN PATIENT 1

MEASUREMENTS		BEFORE OPERATION		FOLLOWING OPERATION
		ANEURISM OPEN	ANEURISM OCCLUDED	
Pulse Rate		80	60	74
Arterial Blood Pressure—Systolic	in mm. Hg.	120	127	115
Diastolic	in mm. Hg.	60	82	72
Venous Blood Pressure	in mm. Hg.	5		
Blood Volume	in c.c.	5400		
Cardiac Output per Minute	in L.	4.50	4.66	4.29
Arm to Face Circulation Rate	in sec.	20	18	19
Mean Velocity of Circulation	in sec.	72	77	
Alveolar Carbon Dioxide Tension	in mm. Hg.	42.3	44.8	44.4
Vital Capacity	in c.c.	4500		4500

Venous Pressure.—The venous pressure, determined according to the technique of Moritz and Tabora,[5] was normal. This is in accord with the findings of Lewis and Drury, and Matas. There have been few other direct measurements in man.

Blood Volume.—The blood volume was obtained by the dye method of Keith, Rowntree and Geraghty.[6] It represented 7.8 per cent of the body weight, which is within normal limits. The effect of a secondary anemia on blood volume is disputed,[7] both high and low values being reported. Further determinations were not performed, since the progressive increase in hemoglobin percentage, which this man showed, would prevent conclusions being drawn from any alteration in the blood volume which might have occurred. Holman[2] has adduced evidence to show that the blood volume is markedly increased in dogs in which arterio-venous anastomoses have been artificially created.

Cardiac Output and Velocity of Blood Flow.—The acetylene method of Grollman[8] was employed in calculating the cardiac minute-volume output. Table II gives in more detail the findings of cardiac output. For the estimation of the cardiac output with the aneurism occluded it was assumed that the oxygen consumption remained essentially unaltered during the time of the experiment. There is some basis for this assumption from experimental work of Lewis and Drury[4] and Harrison, Dock and Holman.[9]

Our results in determining the cardiac output in this subject are not conclusive, except in so far as they indicate that there was no great alteration of the volume flow through the heart. The acetylene method of Grollman which was utilized has been introduced too recently to be established as entirely reliable. Although we have employed it for some months, and our results have been fairly satisfactory, we are not yet ready to draw definite, final conclusions from it. In this particular patient, however, extremely consistent results were obtained. The figures for the cardiac output are within Grollman's normal standards[10] for a man of this size.

TABLE II

MEASUREMENTS OF CARDIAC OUTPUT IN PATIENT 1

DATE	PULSE RATE	ARTERIAL BLOOD PRESSURE		ARTERIO-VENOUS OXYGEN DIFFERENCE C.C. PER L.	OXYGEN CONSUMPTION C.C. PER MIN.	CARDIAC OUTPUT PER MINUTE L.	METABOLISM % OF NORMAL	REMARKS
		SYSTOLIC MM. HG.	DIASTOLIC MM. HG.					
10/29/29	82	120	60	56	260	4.67	+ 4.8	Aneurism open
	60	127	82	55	260	4.70	+ 4.8	Aneurism occluded
10/31/29	80			57	260	4.58	+ 4.8	Aneurism open
	62			56	260	4.62	+ 4.8	Aneurism occluded
12/ 3/29	78			65	281	4.35	+11.6	Aneurism open
12/26/29	82	115	70	70	274	3.90	+10.0	Following operation
				60	274	4.70	+10.0	
1/13/30	67	120	70	67	264	3.96	+ 4.8	
1/17/30	73	110	75	60	235	3.92	- 6.6	
				56	235	4.23	- 6.6	
1/23/30	74			60	282	4.67	+12.3	
				60	282	4.67	+12.3	

It may be claimed that what is measured when the aneurism is patent is not the total cardiac output but that portion of the cardiac output which does not pass through the aneurism. This is true if the blood which passes through the fistula returns to the lungs within the period of the technical procedure. We have reason to believe, from experimental work on the velocity of blood flow, that a relatively small amount of the blood passing through a femoral aneurism will reach the lungs within twenty-five seconds. However, it is probable that our results on the cardiac output with the aneurism open are somewhat too low and that actually the blood flow with the aneurism patent was greater than the flow during compression. Following excision of the fistula there was a tendency for the output to decrease slightly. A complicating factor in this patient was the anemia, for this in itself would tend to increase the cardiac output which would subsequently decrease with the return of the hemoglobin to normal.

Measurements of the velocity of blood flow from the arm to the face as estimated by the histamine method,[11] performed while the aneurism was open and occluded, and also following excision showed no change and were within normal limits.

Measurements were made of the carbon dioxide and oxygen content of arterial and venous blood obtained while the aneurism was open and compressed. The results obtained were too inconclusive to be of value as evidence and have not been recorded. The arterial oxygen capacity of this patient was determined, and rose from an average level of 14.0 volumes per cent before operation to 18.8 volumes per cent three weeks following operation.

(c) *Cardiac Size.*—By percussion and palpation there was questionable cardiac enlargement. A teleroentgenogram of the heart showed slight enlargement to the right, involving the auricle rather than the ventricle, but the total transverse diameter was within normal limits. At fluoroscopic examination, when pressure over the aneurism caused complete occlusion, a slight but distinct enlargement of the right auricle was observed. The border of the auricle moved from three to four millimeters to the right of its previous limits and was maintained there until the pressure was released. This observation was confirmed on repeated examination. One month following operation, the teleroentgenogram of the heart was repeated. At this time the cardiac measurements showed a slight decrease in all diameters from those previously observed.

(d) *Electrocardiogram.*—Electrocardiographic tracings were normal. When repeated with the fistula occluded there was a prolongation of the T-P interval consequent on the slowing of the heart rate, but otherwise no change was to be found, the electrical axis remaining essentially unaltered.

(e) *Skin Temperature.*—Observations of the skin temperature as determined with a thermocouple are recorded in Table III. The right thigh in the region of

TABLE III

SKIN TEMPERATURE MEASUREMENTS IN PATIENT 1

SITE OF MEASUREMENT	SKIN TEMPERATURE READINGS IN DEGREES CENTIGRADE	
	RIGHT	LEFT
Shoulder	32.9	32.9
Abdomen	34.1	34.0
Thigh—above aneurism	34.3	34.7
Thigh—over aneurism	33.9	33.3
Thigh—lateral to aneurism	32.2	32.0
Thigh—medial to aneurism	33.5	33.5
Mid-Thigh	33.8	32.7
Thigh—above knee	33.1	31.9
Lower Leg—below knee	32.9	32.3
Lower Leg—midportion	32.5	31.5
Ankle	33.2	31.5

the aneurism and below it showed a slight tendency to be warmer than the left, and skin temperature measurement over the right lower leg was definitely higher than over corresponding points on the left.

Case II

(a) *Clinical History and Physical Findings.*—This patient (A. C.) was a 25-year-old automobile mechanic whose family and past history was negative. He entered the hospital on October 24, 1929, with the story that five weeks before entry he had been injured in the left wrist by a flying chip of steel. He lost about half a pint of blood before a tourniquet was applied. When this was removed three quarters of an hour later there was no bleeding. The wrist was bandaged for a week and on removing the bandage he noticed a swelling in his wrist which pulsated and gave a thrill. There were no subjective sensations or incapacity, but he had not worked. Physical examination was negative except for the left arm. The lungs showed no abnormality. The cardiac size was well within normal limits and there were no murmurs. At the left wrist over the region of the radial artery, there was an expansile swelling, two centimeters in diameter, over which there was a continuous thrill and murmur with systolic accentuation. For about five centimeters above the swelling the radial artery was very easily palpable, seemed larger than normal, and pulsated more forcibly than on the right. The veins over the internal aspect of the left wrist and forearm were dilated, very prominent, and pulsated, but on raising the arm, collapsed well within normal limits at the same level as those on the right. There were a thrill, pistol shot, and Duroziez's sign over the left brachial artery but not elsewhere. The thrill and Duroziez's sign disappeared instantly on occlusion of the fistula, but the pistol shot persisted though diminished. Capillary pulsation was present to a slight degree beneath the finger nails of the left hand.

Three weeks after entry he was operated upon by Dr. H. B. Loder. A quadruple ligation of the left radial artery and vein was performed, and the aneurismal sac removed. This sac was about two centimeters in diameter with a communication between the artery and vein from one to two millimeters in diameter. Following operation there was no evidence of any circulatory embarrassment to the left hand, but there was an area of partial anasthesia and hyperesthesia over the left thenar eminence. On examination one month following operation he stated that he felt well but had not returned to work. General physical examination was negative. There was no capillary pulse and no pistol shot or Duroziez's sign over any vessel. Both arms appeared to be of equal warmth. There was no atrophy, change in color or loss of power in either arm or hand. At the left wrist on the radial side there was a linear scar five centimeters long which was sensitive to touch. Over the external aspect of the thenar eminence, and extending to include the scar was an area of diminished sensitivity to touch. There was no evident dilatation of the veins of the left arm and hand, and the radial artery above the scar could be felt pulsating and seemed of normal size.

(b) *Circulatory Measurements.*—The pulse rate per minute was 63. When the fistula was compressed there was a slowing to 59. Following operation the pulse rate was 56. The arterial blood pressure in the left arm was 107 millimeters of mercury systolic and 37 mm. diastolic before compression and 110 systolic and 35 diastolic following occlusion. In the right arm the pressure was 100 systolic and 50 diastolic both with the aneurism open and closed. Following operation the blood pressure was 100 systolic and 65 diastolic in each arm. It is notable in this case that there was little systemic alteration in the blood pressure and the general blood pressure was unaffected by occlusion of the fistula. However, the pressure in the artery proximal to the lesion showed a definite lowering of the

diastolic level, and this low diastolic pressure showed no tendency to return to normal when the aneurism was compressed. There must, therefore, have been a decrease in the peripheral resistance in the left arm which was not dependent on the abnormal arterio-venous communication.

(c) *Skin Temperature.*—Measurements of the skin temperature over both forearm and hands are recorded in Table IV. It is to be noted that the skin temperature both proximal and distal to the aneurism on the left was definitely higher than on the right arm. Moreover, the rise in skin temperature was maintained even when the fistula was compressed. This must have been due to an increased blood flow through the arm as a result of a lowered peripheral resistance unrelated to the fistula, in other words, an arteriolar dilatation. One month following operation skin temperature measurements over the two arms were equal.

TABLE IV

SKIN TEMPERATURE MEASUREMENTS IN PATIENT 2

SITE OF MEASUREMENT	SKIN TEMPERATURE MEASUREMENTS IN DEGREES CENTRIGRADE			
	BEFORE OPERATION		FOLLOWING OPERATION	
	RIGHT	LEFT	RIGHT	LEFT
Above Elbow—lateral	31.4	33.5	29.2	29.2
Below Elbow—medial	29.9	34.2	29.9	30.2
Mid-Forearm—lateral	29.5	31.3	29.5	29.4
Mid-Forearm—medial	29.8	34.1	29.9	30.4
Wrist—lateral (over aneurism on left)	29.7	34.3	30.9	29.5
Wrist—medial	28.7	33.3	31.1	31.7
Thenar Eminence	28.8	31.6	30.3	29.7
Hypothenar Eminence	27.8	31.6	31.1	31.4
Palm of Hand	29.5	32.7	31.4	32.0

(d) *Tourniquet Test.*—The total blood supply to the left arm was suddenly occluded while the arm was held upright to facilitate venous return. The arm was then lowered and with the fistula compressed immediately the tourniquet was released. A sudden and intense flush appeared over the ulnar and median aspects of the hand and forearm. The radial aspect of the hand became pink more slowly. When the radial artery was released, the thenar eminence immediately became pink.

When the test was repeated over the right arm, the color returned to the arm and hand more slowly and less intensely than it did on the left, a fact which the patient noticed and commented upon. Since the returning inflow of blood was able to reach the minute skin vessels of the left arm very much more quickly than those of the normal arm, this test, too, suggests that there must have been an arteriolar relaxation in the left arm. Moreover, this dilatation was unaffected by mechanical occlusion of the fistula.

(e) *Carbon Dioxide and Oxygen Content of Arterial and Venous Blood.*—The content of carbon dioxide and oxygen from the antecubital veins of both arms and of arterial blood is recorded in Table V. The blood from the left antecubital vein closely approached the arterial blood in character, whether the aneurism was patent or occluded. To obtain the sample following occlusion the arm was first held upright for one minute after compression of the aneurism in order to drain the blood from the veins. This experiment was repeated subsequently with identical results. Since there is no reason to believe that the metabolism of the two arms differed, the decreased oxygen utilization in the left arm must have been due to an increased blood flow in this region. Following operation the venous blood from both arms showed a normal content of carbon dioxide and oxygen.

TABLE V

DETERMINATION OF THE CARBON DIOXIDE AND OXYGEN CONTENT OF ARTERIAL AND VENOUS BLOOD FROM PATIENT 2

BLOOD FROM	PRE-OPERATIVE		4 WEEKS POST-OPERATIVE	
	CARBON DIOXIDE VOL. %	OXYGEN VOL. %	CARBON DIOXIDE VOL. %	OXYGEN VOL. %
Left Antecubital Vein—aneurism open	47.76	18.96		
Left Antecubital Vein—aneurism occluded	47.68	18.75	54.72	12.82
Right Antecubital Vein	54.39	12.24	55.62	12.65
Artery	47.28	19.51		

To summarize, evidence from four sources has been presented in this case of a lowering of the peripheral resistance in the neighborhood of the arteriovenous fistula. This lowered peripheral resistance is not due directly to the arterial leak, but to a generalized relaxation of the vessels where the resistance is normally highest; in other words, an arteriolar dilatation. The cause of the production of this dilatation will be considered later.

DISCUSSION

These two cases are of interest because, although both presented the characteristic local signs of an abnormal arterio-venous communication, they were dissimilar as regards the systemic circulatory manifestations. Matas[3] cites seven factors which are of importance in determining the effect of the fistula on the general circulation. These are: (a) the size of the fistula; (b) the volume and force of the arterial stream which is short-circuited; (c) the calibre of the vessels involved; (d) the proximity of the fistula to the heart; (e) the duration of the condition; (f) the age of the patient; and (g) the presence or absence of co-existent heart disease. In both cases here reported, the patients were young with previously normal cardiovascular systems and the fistulae were of short duration. However, in the first subject the fistula was of somewhat greater size than in the second, the volume of blood short-circuited was presumably greater, and the vessels involved were the femoral, of large calibre and relatively close to the heart, instead of the more peripherally situated radial vessels in the second patient. It is, therefore, natural to expect that the first man should show definite and the second very slight systemic effects.

Heart Rate.—A slowing of the heart rate upon compression of the aneurism was observed in both patients, although only slightly in the second case. Lewis and Drury[4] found that this effect was abolished by atropinization, and, therefore, attributed it to a vagal reflex initiated by the rise in mean arterial pressure. Rieder,[12] however, was able to elicit the slowing even after the injection of atropine, although the dosage which he employed was only half that given by Lewis. Rieder was also able to obtain the bradycardiac effect in dogs following division of the vagi. It is possible that there may be cardio-inhibitory

fibers, parasympathetic in nature, other than those which are contained in the vagus nerves. In the light of the recent work on the importance of the carotid sinus in the regulation of heart rate and blood pressure by Hering,[13] Heymans[14] and others, it is possible to advance two theories to account for the production of this reflex. First, the sudden increase in mean arterial blood pressure which occurs upon occlusion of the fistula may initiate the reflex in the carotid sinus itself. Second, the aneurism itself may locally assume a regulatory function such as is known to be possessed normally by the carotid sinus, and, upon the sudden alteration of the pressure in it or over it, be the starting point for a nervous reflex which terminates in cardiac inhibition. The pathway through which this reflex may travel is not clear. It has been shown[15] that spinal anesthesia does not abolish the phenomenon.

Cardiac Output.—Lewis and Drury[4] claim that there is no significant increase in cardiac output in the presence of an arterio-venous aneurism. They base this assumption on their inability to find an increase in general venous pressure, and on the experimental production of fistulae in dogs in which, in the absence of a rise in venous pressure, they were unable to detect any appreciable change in the cardiac output per minute. The mere fact that there is no increase in venous pressure appears to be an inadequate criterion for concluding that the volume flow through the heart is unaltered. While the systolic output does indeed depend on the venous return to the heart, it is possible that a heart with normal functional capacity responds so immediately to a relatively small augmentation of the venous return that it is impossible to detect clinically any rise in venous pressure. Indeed, clinical observations in certain conditions where the cardiac output is known to be increased, for example in hyperthyroidism, frequently fail to reveal an increased venous pressure. In arterio-venous fistula, it would appear quite possible for the cardiac output to increase without a demonstrable increase in venous pressure, provided the heart functions normally and the lesion is of sufficient duration to enable the circulation to have adapted itself to the altered conditions.

As opposed to the findings of Lewis and Drury, Harrison, Dock, and Holman[9] have presented evidence of an increase in cardiac output in dogs in which arterio-venous anastomoses had been produced experimentally. In their work the venous pressure was not measured, and it must be appreciated that the fistulae which were created were of a relatively large size.

In Case I no change in the cardiac output per minute was found when the fistula was acutely compressed, although for reasons already discussed these measurements may not have represented the total volume flow. Following operation when the circulation had readjusted itself to normal conditions, the volume flow showed a tendency to decrease somewhat, but the decrease in the anemia in itself might have been a

factor in the decrease in heart output. No conclusive evidence was obtained, therefore, that in this particular patient the arterio-venous aneurism increased the cardiac output greatly. In other patients with aneurisms of more considerable extent it is reasonable to believe that the heart output may show a marked rise. It is also probable that the cardiac output in cases of arterio-venous aneurism may be influenced according to whether the patient performs a normal amount of work, or as in Case I, the patient is resting in bed following the development of the fistula.

Peripheral Resistance.—From anatomical and physiological considerations, one cause for the lowered peripheral resistance occurring in arterio-venous aneurism can be found in the direct short-circuiting of the blood stream from an artery to a large vein. This must be so because not only are the physiological manifestations of a lowered resistance, particularly a reduced diastolic blood pressure, approximately in proportion to the extent of the communication, but many of these abnormal phenomena can be immediately abolished by compression of the fistula.

From the studies made on our second case, however, definite evidence was obtained that there was a lowering of the peripheral resistance, unconnected with the actual fistula, in other words, an arteriolar relaxation. The exact nature of the production of the dilatation cannot be explained conclusively, but it is certain that it is related in some way to the abnormal circulatory conditions consequent on the arterial leak. There are certain possible factors, any one or a combination of which may be etiologically responsible. First, there is the local damage to the vessel wall itself in the fistula as well as the widening of the artery proximal to it. These factors seem unlikely as the cause when one considers the extensive vascular damage, particularly in pure arterial aneurisms, which may occur without reflex arteriolar dilatation. Second, the stretching and engorgement of the veins communicating with the fistula might initiate such a reflex. When veins are thus engorged with blood under a relatively high pressure there is a retrograde pressure in the capillaries and a tendency for capillary blood flow to cease. Reflexly, then, the arterioles might dilate in an endeavor to maintain the normal capillary circulation. A third explanation, also, can be given for the diffuse arteriolar relaxation. The increased pulse pressure, with its resulting lowering of the mean arterial pressure, which has already been produced by the fistula, may produce the arteriolar dilatation in an endeavor to maintain the capillary pressure at the normal level which is essential for the adequate exchange of gases between tissues and blood. These possible mechanisms of the production of the lowered peripheral resistance have been presented as theories only. The question is an open one and awaits further investigation.

It is possible that this regional vascular dilatation may play a rôle in what is known as "the establishment of collateral circulation." In

particular, this phenomenon may serve a purpose in maintaining an adequate blood flow to the tissues until the time when the regional vessels have become permanently enlarged. We have shown that an arteriolar dilatation may occur in conjunction with arterio-venous aneurism. In cases showing fistulae of greater size, may this arteriolar dilatation be more extensive, even generalized, and contribute to some of the systemic effects which are observed? The problem of attempting to demonstrate this is complicated by the fact that a direct lowering of the peripheral resistance through the arterio-venous shunt is a very important, if not the major, factor in producing the systemic manifestations. Any attempts to exclude this factor by temporary compression of the aneurism result in a serious altering of the physiological conditions from those to which the circulation has already become adapted by redistribution of the blood volume, changes in the cardiac output, etc. For this reason, the influence of an arteriolar dilatation may not be readily discernible. One phenomenon, however, which is commonly seen in cases of arterio-venous fistula, and which is uninfluenced by temporary compression of the communication, is that of "capillary pulsation." This was observed in our first patient, and was also commented upon by Lewis and Drury.[4] Lewis[16] has recently presented definite evidence that this phenomenon is in truth occasioned by an arteriolar dilatation. If, therefore, capillary pulsation does indicate an arteriolar relaxation, as seems highly probable, there are excellent grounds for the belief that an arterio-venous aneurism may produce a generalized arteriolar dilatation as well as the local dilatation which we have shown does occur.

It is quite possible, moreover, that such a reflex lowering of the peripheral resistance may exist in certain other clinical conditions in which the circulatory manifestations are somewhat similar to arterio-venous aneurism. In particular, this may be true in aortic insufficiency. Lewis and Drury concluded, as a result of their investigation of arterio-venous aneurism, that in aortic regurgitation, the amount of blood regurgitating into the left ventricle must be a significant quantity. This is in opposition to the view first advanced by Stewart[17] that the quantity of regurgitating blood is trivial and that the peripheral circulatory manifestations are the result of a reflex lowering of the peripheral resistance. As stated previously, we believe that the criteria upon which Lewis and Drury base their assumption that the cardiac output in arterio-venous fistula is unaltered are inadequate, and we also believe that there is evidence, both theoretical and experimental, that the cardiac output may be increased in arterio-venous fistula. Our investigation has thrown no light on the question as to whether or not the amount of regurgitating blood in aortic reflux is significant. However, since evidence has been presented by us that a generalized lowering of the peripheral vascular resistance may exist in cases of arterio-venous

aneurism of moderate size, it is very suggestive that in aortic insufficiency, which presents analogous peripheral circulatory phenomena, including capillary pulsation, there may also be a diminution of the peripheral resistance.

To sum up: an arterio-venous aneurism produces characteristic local signs and symptoms which are too well known to detail here. It also may be responsible for certain general effects on the circulation, which vary in their occurrence and extent with the size and location of the fistula. The most characteristic of these phenomena are, (a) an increase of the heart rate, with an immediate slowing upon compression of the aneurism; (b) a decrease in the diastolic arterial blood pressure, with a resulting increased pulse pressure; (c) cardiac enlargement; (d) a redistribution of the blood volume with a tendency toward an accumulation of blood in the venous portion of the vascular circuit, and probably an increase in the total blood mass; (e) a normal or possibly increased cardiac output, depending on the degree of fistula; (f) a regional and frequently a generalized arteriolar dilatation.

In our first patient, the general effects of the lesion were apparent but not marked. This may have been due to the comparatively small size of the fistula. However, it was 3-4 millimeters in diameter, which is as large as the internal diameter of the brachial artery, and must have conveyed a considerable amount of blood. The fact that this man was at absolute rest between the time of his injury and operation may explain to a certain extent the lack of more marked systemic involvement. We believe that patients with arterio-venous fistulae showing such systemic effects should have the benefit of as complete rest as possible in order to spare their general cardiovascular system, and that operative excision of the lesion should be performed in every case without undue delay.

SUMMARY

1. Two cases of traumatic arterio-venous fistula were studied before and after surgical operation. Both patients showed the characteristic local signs of the condition, and the first patient presented the classical phenomena of the effect on the general circulation.

2. In the first patient estimations of the cardiac output were performed, and although the applicability of the method is somewhat limited in this condition, the measurements showed no marked change upon compression of the aneurism and a slight tendency for a decrease following operation. The velocity of blood flow in this subject showed no change at any time. The blood volume was normal.

3. Evidence was presented of an arteriolar dilatation in the surrounding region of the arterio-venous aneurism in the second patient.

4. It was suggested that in certain cases of arterio-venous aneurism of marked extent, the arteriolar dilatation may be generalized. Likewise, in aortic insufficiency such an arteriolar dilatation may exist. This

would explain the capillary pulsation and to a certain extent other of the peripheral circulatory phenomena which are observed both in arterio-venous fistula and aortic regurgitation.

We take pleasure in expressing our appreciation of Miss Rose Shore's technical assistance in conducting this research.

REFERENCES

1. Reid, Mont R.: The Effect of Arteriovenous Fistula Upon the Heart and Blood Vessels. An Experimental and Clinical Study, J. H. H. Bull. **31**: 43, 1920.
2. Holman, Emile: Experimental Studies in Arteriovenous Fistulae. I. Blood Volume Variations. II. Pulse and Blood Pressure Variations. III. Cardiac Dilatation and Blood Vessel Changes, Arch. Surg. **9**: 822, 1924.
3. Matas, Rudolph: On the Systemic or Cardiovascular Effects of Arteriovenous Fistulae, Internat. Clinics **2**: (Series 35), 58, 1925.
4. Lewis, T., and Drury, A. N.: Observations Relating to Arteriovenous Aneurism. I. Circulatory Manifestations in Clinical Cases with Particular Reference to the Arterial Phenomena of Aortic Regurgitation. II. The Immediate Effects of an Arteriovenous Anastomosis on the Dog's Circulation, Heart **10**: 301, 1923.
5. Moritz, F., and Tabora, D. V.: Über eine Methode beim Menschen den Druck im oberflächlichen Venen exakt zu bestimmen, Deutsche Arch. f. klin. Med. **98**: 475, 1910.
6. Keith, N. M., Rowntree, L. G., and Geraghty, J. T.: A Method for the Determination of Plasma and Blood Volume, Arch. Int. Med. **16**: 547, 1915.
7. Rowntree, L. G., Brown, F. E., and Roth, G. M.: The Volume of Blood and Plasma in Health and Disease, Philadelphia, 1929.
8. Grollman, A.: The Determination of the Cardiac Output of Man by the Use of Acetylene, Am. J. Physiol. **88**: 432, 1929.
9. Harrison, T. R., Dock, W., and Holman, E.: Experimental Studies in Arteriovenous Fistulae. Cardiac Output, Heart **11**: 337, 1924.
10. Grollman, A.: Physiological Variations in the Cardiac Output of Man. VI. The Value of the Cardiac Output of the Normal Individual in the Basal, Resting Condition, Am. J. Physiol. **90**: 210, 1929.
11. Weiss, Soma, Robb, G. P., and Blumgart, H. L.: The Velocity of Blood Flow in Health and Disease as Measured by the Effect of Histamine on the Minute Vessels, Am. Heart J. **4**: 1, 1929.
12. Rieder, W.: Herzschädigung infolge arterio-venösen Aneurysmas, Arch. f. klin. Chir. **129**: 597, 1926.
13. Hering, H. E.: Die Karotissinusreflexe auf Herz. u. Gefässe, Dresden, 1927.
14. Heymans, C.: Le Sinus Carotidien, Zone Réflexogène Régulatrice du Tonus Vagal Cardique du Tonus Neurovasculaire et de l'Adrénolinosecrétion, Arch. Internat. de Pharmacodynamie et de Thérapie. **35**: 269, 1929.
15. Gerlach, F., und Harke, W.: Ein Beitrag zur Frage der Entstehung der Blutdrucksteigerung und Pulsverlangsamung bei Kompression arteriovenöser Aneurysmen, Klin. Wchnschr., **3**: 980, 1924.
16. Lewis, Thomas: The Blood Vessels of the Human Skin and Their Responses, London, 1927.
17. Stewart, H. A.: Experimental and Clinical Investigation of the Pulse and Blood Pressure Changes in Aortic Insufficiency, Arch. Int. Med. **1**: 102, 1908.

CARDIAC SYMPTOMS NOT DUE TO CARDIAC DISEASE

HENRY FARNUM STOLL, M.D.
HARTFORD, CONN.

CONFUSED and fantastic though the layman's knowledge of anatomy may be, he invariably knows where his heart is located and with rare exception, attributes every unusual sensation in this region to disease of that viscus. The fact that not infrequently it is impossible to detect serious disease of the heart even with the aid of instruments of precision, justifies the apprehension that pain in this region occasions.

I have recently had the opportunity of studying two patients presenting cardiac symptoms due to extra-cardiac causes that puzzled me greatly for some time. In one, pain over the precordium was the chief complaint; the other suffered from attacks of palpitation, the cause of which was only revealed when the early emotional life of the patient was carefully investigated.

CASE 1.—D. D., an unmarried woman, aged 25 years, who was employed painting dials on clocks with radium, was admitted to the Hartford Hospital October 8, 1928, complaining of pain about the heart and stomach. The former pain had been present several years and was the more severe. It would come on suddenly, last an hour or all day and would not radiate. It was located internal to the cardiac apex. The other pain was behind and below the lower end of the sternum and was not affected by food but was sometimes relieved by alkalies which, however, never relieved the precordial pain. The patient had lost considerable weight and suffered somewhat from dyspnea and palpitation.

The important physical findings and laboratory data were as follows: She was a tall slender girl with marked reduction of antero-posterior diameter of the chest; slight enlargement of thyroid isthmus; heart not enlarged, but a loud harsh systolic murmur was heard all over the precordium, loudest internal to the apex.

Left knee reflex not obtained; right elicitated only with reenforcement: Hb., 64 per cent; R.B.C., 3,420,000; W.B.C., 6100 (64 per cent lymphocytes); blood culture, Wassermann test and stool all negative. Roentgenography of the gastro-intestinal tract was negative for ulcer but showed a slight six-hour stasis. Basal metabolism rate was −10 per cent; temperature and pulse were normal. Notwithstanding a negative history of rheumatism, I feared that she probably had an old rheumatic mitral valve lesion and that the pain probably indicated activity. The pain behind and below the lower part of the sternum was at first thought to be probably due to a peptic ulcer even though this diagnosis was not substantiated by the radiological examination. Both of these assumptions proved wrong.

One consultant thought that the pain was perhaps due to pressure from the thyroid. When first seen, the possibility of poisoning from radium was uppermost in our minds. There was moderate secondary anemia but a normal leukocyte and differential count. A few months later, Dr. Frederick B. Flynn, of Columbia University, gave her the electroscopic and expired air tests, which will detect as small

an amount as two micrograms of radium, with negative results. The change in the knee reflexes was of temporary nature.

The precordial pain promptly subsided upon rest in bed and the institution of the Sippy régime for ulcer seemed to control the epigastric pain but when the patient began to get up after three weeks, preparatory to going home, both pains recurred as severely as before. Our mutual disappointment was great. I then did what I should have done three weeks previously, examined her in a standing position. She stood with markedly drooping shoulders, protuberent abdomen and greatly exaggerated lumbar lordosis. When sitting, she slouched with shoulders forward. Believing that the pain might be the result of faulty posture either from radicular irritation or pinching of the intercostal nerves by approximation of the ribs anteriorly, her shoulders were strapped back with adhesive plaster and the abdomen supported in like manner. This assumption proved to be correct as she was at once relieved from pain. Inquiry then revealed that in the factory she worked at a low table and as she was above the average height, she had to sit stooped forward all day. She was instructed to bend forward from the hips, keeping the spinal column straight. Upon her return to the factory, the worktable was raised and she has now worked several months without pain. Once or twice when there was a suggestion of the old pain, she stopped it at once by correcting her posture.

COMMENT

The patient's disability was occupational in nature; not, however, due to the fact that she was using radium but to the position she assumed while using it. Had the patient been examined in the office, her bad posture would undoubtedly have been detected at the first consultation but as she was in bed when first seen, she was not examined standing until several weeks had passed. No patient can be considered to have had a complete examination until observed in the sitting and standing positions. If the patient be a machine operator for instance, a great deal of light is often thrown on an obscure pain if one observes the posture assumed while he performs the various motions used in operating the machine. Within a few weeks, a slender girl who worked as a cashier, consulted me because of precordial pain especially marked in the late afternoon. No organic cause was found but it was readily apparent her posture was very poor. The cause of the pain was explained to her and she was instructed how to maintain a correct posture. One week later she said she had had no recurrence of the pain. Several similar cases have been seen. Neither Levy[1] nor White and Wood[2] in their recent contributions on cardiac pain, refer to bad posture as a cause. The latter authors, speaking of simple fatigue pain state that "It may be the result of a number of different factors." Gunther and Sampson,[3] in discussing the pain referred to the precordial region, due they believed to radicular irritation the result of hypertrophic spondylitis, state, "The intensity of symptoms does not parallel the degree of anatomic changes shown by roentgen examination. It is evident that their symptoms depend not only on the mechanical factors mentioned but on others which at present are not so clear."

It would seem that the mechanical factors might be especially prone to be operative when the upper segment of the spine is strongly flexed, as occurs with the slouching posture. Hypertrophic spondylitis is rare in early life but common past middle age. Garvin[4] in the study of 2,000 radiograms at the Mayo Clinic found evidence of hypertrophic spondylitis in 67 per cent of men and 40 per cent of women over fifty but in 74 per cent of the former and 61 per cent of the latter it appeared to cause no symptoms.

Gunther and Sampson noted in their patients that the pain might be absent on arising in the morning, due they believed to increase in the tonus of the paravertebral muscles. Obviously, the effect of the increase in muscle tone enables the individual to hold himself more erect and consequently he has less pain. The return of the pain on prolonged sitting must be due to the increase in the forward bending of the spine as the individual slumps. Adhesive strapping to hold back the shoulders for a week, instruction how to walk and sit, together with exercise to develop the spinal muscles will promptly effect a cure if posture be the cause.

Case 2.—Viola B., aged 27 years, married, was admitted to the Hartford Hospital because of attacks of palpitation. The attacks began in childhood and have occurred especially at night. With these attacks she "gets cold all over" and her heart "pounds" very hard. They come about once in two or three weeks though for a time when the patient was about 16 years old, they were almost daily. Seven years ago she married and went to Florida to live and while there, the attacks were very rare—only once a year. She left her husband one year ago because of his bad habits. Two children are living and well. For the past two years she has spent the summer north with her parents and at these times the attacks have been much more frequent, often awaking her in the night. She feels "dopy" and is tired after them but has no pain at any time. As she has to earn her living, the attacks greatly interfere with her work and she fears she has heart trouble.

She was an attractive young woman who, aside from being slightly undernourished, had a negative examination except for a rather low metabolism rate (−19 per cent). Thyroid administration did not improve the situation. At first I attributed the attacks to her separation from her husband, especially as they were rather more often at night. "How do you explain the fact" said she, "that I lived without him for a year in Florida with only a very rare attack and have had frequent attacks when living with my father and mother who try to do everything for me"? It was some time before I could answer her question and though I told her they were not serious, I could not convince her until I was able to explain them.

Was the increase in the number of attacks when with her parents an indication of a desire on her part for attention and solicitude? She was an only child and though very attractive physically, she had not been able to hold her husband's affection. Did the attacks signify a "bid" for parental solicitude?

It is quite common to see illness used as a means of obtaining attention and this possibility might explain the present attacks but would they explain those that occurred in early childhood?

As they began when she was seven years old, I finally asked her in detail about her childhood. I learned it had not been happy because of her father's alcoholism. He would get very drunk and be very abusive. He frequently threw dishes about,

shouted in loud tones and threatened to kill both mother and daughter. She would often go to bed too frightened to sleep and at these times, when she thought he might be coming in to carry out his threat, the attacks of palpitation had their inception. Her father reformed many years ago and the family is now a happy one but the patient said when she recently returned home she could hardly bear the sight of the household furnishings. Her grandmother's picture hung on the wall: she had no ill feelings against her but it looked down at her years ago when she was a terrified child and she dreads to look at it now: so it was with some of the dinner dishes. It seems as if she could not eat from the dishes remaining from the set from which her father had selected missiles to terrify the family in the years gone by. "I told mother," said she, "I believed I would be better if we got rid of all the old things." I suggested that as her father was no longer a drunkard, the furnishings of the house should remind her, not of the terrifying scenes of childhood but of the victory her father had won. She was further told that as she had had so many attacks in childhood, the mechanism that started them became so sensitive that it would sometimes be set going without any discernible cause. This explanation seemed to appeal to her and the attacks of palpitation became much less frequent and did not cause her the anxiety they had formerly occasioned.

COMMENT

This case illustrates how important it is to secure emotional data in obtaining a medical history. It is probable that "painful memories, present dilemmas and fears regarding the future" (Favill) cause as much suffering as, if not actually more than, organic disease. The sympathetic physician who takes the necessary time to study the emotional background of his patients is amply rewarded for his pains.

SUMMARY

Symptoms referable to the region of the heart, especially occurring in early life, are often not due to cardiac disease. Two illustrative cases are here presented. In one—pain was due to faulty posture assumed at work; in the other attacks of palpitation owed their inception to a long-continued state of terror in early childhood.

REFERENCES

1. Levy, Robert L.: Amer. Heart Jour. **4**, 377, 1929.
2. White, Paul D., and Wood, J. Edwin: J. A. M. A. **81**, 539, 1923.
3. Gunther, Lewis, and Sampson, John J.: J. A. M. A. **93**, 514, 1929.
4. Allard, Louis W.: J. A. M. A. **93**, 1556, 1929.

HEART DISEASE IN THE STATE OF NEW YORK. A STATISTICAL REVIEW OF MORTALITY AND MORBIDITY*

J. V. DePorte, Ph.D.
Albany, N. Y.

OUR mechanical age is a rapid succession of conquests over nature. The plots of Old World fairy tales and of dime novels in this country, in which a thing is "no sooner said than done," have become in the popular philosophy of the day, almost a reality. In our pride over material gains, we are in danger of overlooking the permanent values of human progress. Even some of our universities not infrequently speak in the language of real estate promoters and business boosters. While money undoubtedly facilitates scientific research, one must remember that there is no direct relation between the sums expended and the degree of success of the research. The scientific worker must guard against the prevalent conception of life. The motivating force in his work should be an urge for truth irrespective of its immediate or even ultimate utilization.

Although vital statistics is largely a practical method, a certain degree of detachment in the collection of the data and formulation of conclusions is, in my opinion, as essential here as it is in the pure sciences. The field of vital statistics is practically limitless. Everything and anything that affects the life and physical well-being of individuals and communities usually can be and frequently is expressed in numerical terms, out of which are built the prosaic-looking statistical tables. Only one or two generations ago statistical expressions were frequently guesses, or at best, rough approximations to reality. In those days, when some cities took great satisfaction in announcing death rates of 1 or 2 per thousand, and in one historic instance of a fraction of a point, even a poorly equipped but earnest prospector had comparatively little difficulty in uncovering nuggets of fact. We have now reached a stage where the search for truth requires more concentrated thought and greater refinement of tools. We must sink our wells deeper, we must work ores whose yield is little when compared with earlier and more prosperous days.

MORTALITY FROM HEART DISEASE

Of late years there has been considerable alarm over the high mortality from heart disease and especially its upward trend. If figures were infallible the alarm would be only too natural. In the State of

*Read before the American Heart Association, New York City, February 3, 1930
From the Division of Vital Statistics, New York State Department of Health.

New York, for example, heart disease has been the leading cause of death since 1912, with the single exception of 1918, when pneumonia held first place with a death rate of 337.5 per 100,000 population, followed by influenza (259.1); heart disease being third (239.0). In 1928 the death rate from heart disease was the highest ever recorded (297.9) and the deaths ascribed to this group of causes were responsible for almost one-fourth of all deaths (22.8 per cent).

The importance of heart disease as a cause of death increases with age. In 1928, in the entire State, it was sixth among the causes of death of children under 10 years of age who had weathered the first year of life, pneumonia being first, followed by diphtheria, tuberculosis, diarrhea, and measles. It was second in the age group 10-20 years, preceded only by tuberculosis, and third between the ages 20-40 years, tuberculosis being first and pneumonia second. After the 40th year, heart disease held first place.

Of the 34,597 deaths from heart disease, 41.1 per cent occurred after the 70th year, 50.6 per cent between the ages of 40 and 70, and only 8.3 per cent of the deaths were among persons under 40 years of age.

The curve of mortality from the beginning of the century has had an almost continuous rise from a rate of 133.5 in 1900 to 297.9 in 1928. There were only five recessions in this twenty-nine year period, the greatest of which occurred in 1919, when, as a sequel of the influenza epidemic the contingent of sufferers from impairments of the heart was considerably reduced. There is no unanimity among physicians and statisticians regarding the significance of this increase. In fact, some of them believe that the increase is more apparent than real. They stress the fact that the population of this country has been growing older, both because of the control of certain important causes of death in infancy and childhood and the resulting increase in the contingent of older persons, and in recent years the reduction in the volume of immigration which had given us hitherto an abnormally large proportion of young people. The mortality from heart disease being confined largely to the older ages, with an increase in the proportion of persons in those ages, the death rate naturally rose. Then we are told that improvements in diagnosis and the more careful statements of the cause of death, which has practically eliminated the rubric "old age" and reduced considerably deaths from causes "unknown," have also contributed to an increase in the recorded total of deaths from heart disease.

Let us now consider briefly some statistical sources of error in the recorded rates. There is, first of all, the fact that the statistical procedure as outlined in the International List of Causes of Death is revised every ten years, thus affecting the comparability of the rates. Of greater importance is the generally employed method of selecting the

cause to which a death should be assigned when a doctor enters two or more causes on the certificate. In 1925, for example, in the State, outside of New York City, almost one-half of the death certificates showed more than one cause. The selection is, of necessity, guided by the rather arbitrary set of rulings of the *Manual of Joint Causes of Death.* These instructions are at times contrary to the opinion of the certifying physician. In order to determine to what extent the occasional disagreements between the opinion of the physician and the statistical procedure influence the recorded death rates from heart disease, we have examined the certificates which were filed with the State Department of Health in March, 1928, in which heart disease was shown either as the primary or secondary cause. The results are shown in Table I.

TABLE I

DEATHS FROM DISEASES OF THE HEART BY AGE—MARCH, 1928, NEW YORK STATE (EXCLUSIVE OF NEW YORK CITY)

AGE	EDITED ACCORDING TO INTERNATIONAL LIST OF CAUSES OF DEATH AS:		CERTIFIED BY PHYSICIAN AS:	
	PRIMARY	SECONDARY	PRIMARY	SECONDARY
All ages	1,551*	635	1,451	735
Under 5 years	14	24	16	22
5- 9 years	3	8	6	5
10-14 years	5	4	6	3
15-19 years	5	8	9	4
20-24 years	8	9	8	9
25-29 years	13	18	18	13
30-34 years	18	18	22	14
35-39 years	25	16	30	11
40-44 years	40	24	38	26
45-49 years	49	38	51	36
50-54 years	86	45	80	51
55-59 years	122	52	120	54
60-64 years	160	71	149	82
65-69 years	193	83	176	100
70-74 years	225	76	209	92
75-79 years	246	59	218	87
80-84 years	198	52	174	76
85-89 years	100	20	86	34
90-94 years	33	9	30	12
95 years and over	8	1	5	4

*Including 13 delayed certificates for deaths which occurred in February.

The number of certificates totalled 2,186. In 1,451 of these, the physicians entered heart disease as the primary cause while, when edited according to the *Manual*, cases of primary heart disease numbered 1,551—an excess of 100, or 6.9 per cent.

The recorded number of deaths under 40 years totalled 91, the corresponding number assigned by physicians being 115. In other words, if the statements of the physicians were taken in all cases the death rate under 40 in March, 1928, would have been greater than the re-

corded rate by 26 per cent. In the older ages the reverse was true. At 40-60 years the recorded rate was 2.8 per cent higher and in the group 60 years and over it was 11.1 per cent higher than the rate based on the totals of primary cases assigned by the physicians.

The figures illustrate the degree of error arising out of the statistical procedure governing the tabulation of deaths according to cause. Although, to be sure, they represent the experience of only one month, the disparity between the recorded figures and the entries of the physicians would generally hold true for any month.

When we say, therefore, that the death rate from heart disease in the State, outside of New York City, was 308.3 in 1928, this number by no means measures accurately the mortality from this cause in the sense in which the reading of a clinical thermometer measures a patient's temperature. The difference between the present death rate and the rate, say, in 1900 is a still less accurate measurement of the change in the mortality during the twenty-nine year period.

Should one, therefore, forego an analysis of mortality from this and from certain other causes because of the large error contained in the figures, as in fact an eminent pathologist wrote to me some time ago? The answer to this question is, clearly, *no*. We cannot sit idle and blissfully await the day when the entire truth will be disclosed to us. A death rate is in a sense a theory which is constantly being made more accurate by the greater accumulation of knowledge.

While we do not know the exact number of deaths in which heart disease is the deciding factor, we do know that it is large. In 1928 almost thirty-five thousand deaths in the State were assigned to heart disease as the primary cause. If we were to consider the certifications of physicians and assume the degree of discrepancy deduced from the March, 1928 figures, the number would be reduced by 7 per cent to about thirty-two thousand. Although it is impossible to make a correction for errors in diagnosis, the number gives us a true picture of the relative position of heart disease among causes of death.

The mortality in old age, after the 70th year when more than two-fifths of the deaths occur, cannot be reduced to any appreciable extent since most of it is merely the natural result of the wearing out of vital organs due to the mere process of living. It is the younger ages that should be the concern of the physician and public health worker. In 1928 the deaths of 2,867 persons under 40 years of age were ascribed to heart disease. This is a larger number than the deaths at all ages during that year from diphtheria (863), influenza (1,943), all puerperal causes (1,295), and almost equal to the mortality from diabetes (3,069). There were recorded 2,927 deaths from heart disease in the age group 40-50 years, 5,630 deaths at 50-60 years, and 8,944 at 60-70 years.

The question naturally arises—can the mortality from heart disease in the younger ages be reduced and by what means? No satisfactory

answer to this question can be made without definite knowledge of the prevalence of heart disease, of its etiology, the present methods of treatment, and their effect. Here, preventive and curative medicine could make the best use of adequate morbidity statistics if they were available.

Official health agencies do not require the reporting of heart disease, the State and city sanitary codes limiting their lists to those diseases which are likely to spread from individual to individual. In this day, when the immutability of even chemical elements is no longer an axiom, the rigid grouping of diseases into communicable and non-communicable seems to be altogether artificial. Broad considerations of public health necessarily lead one to inquire into all causes of ill health. The existing system of disease reporting gives us only a partial answer.

Several years ago it occurred to us that it might be possible to interest a sufficient number of physicians in a coöperative study of morbidity of the "non-communicable" type. Although this expectation was contrary to what is supposed to be the practical American's credo, that "you cannot get something for nothing," I have always been convinced that most of us have an urge to spend some of our time in doing what we feel is worth while and for which we can expect no compensation other than the approval of our inner selves and the possible approbation of our neighbors.

The plan for a year's survey of sickness in the rural part of the State (to which I shall refer henceforth as the "Rural Survey") was presented in the summer of 1925 to a gathering of physicians and later published in *Health News*, the official organ of the Department. The response was most gratifying; more than one hundred physicians in thirty-three counties consented to make weekly reports on forms furnished by the Department of the occurrence in their practices of certain non-reportable diseases. This survey was carried out during the year 1927.

Later, it was decided to attempt a similar but more intensive survey of a single county and Essex was chosen for this purpose. The second survey, in which twenty physicians participated, continued for fifty-two weeks, October 2, 1927-September 29, 1928.

Let me summarize briefly the results of these surveys, especially those bearing on the subject of this paper. The total number of cases reported in the Rural Survey* was 98,069. Cases of heart disease numbered 4,123. In terms of the population (which was estimated as one hundred thousand), this indicates that about 4 per cent were suffering from some form of heart disease.

*Sickness in Rural New York. J. V. DePorte, Journal of the American Medical Association, Vol. 92, pp. 522-528.

This proportion is undoubtedly somewhat of an exaggeration since the forms on which the reports were made do not give the names of patients and it is possible that some cases may have been reported by more than one physician. In rural districts, however, the practice of changing physicians is not common and the degree of error introduced by repetition could not be considerable.

There were also reported 6,038 cases of tonsillitis, 3,234 of arteriosclerosis, 2,240 of chronic arthritis, and 1,543 of acute rheumatic fever.

In the Essex Survey* the total number of cases reported was 19,179. The form employed differed somewhat from that used in the Rural Survey, a new rubric "diseases of the skin" being added; syphilis subdivided into acquired and congenital, and diseases of the heart (this at the suggestion of Dr. R. H. Halsey), into rheumatic, syphilitic, and "other forms." Cases of heart disease numbered 535, or 3.0 per cent of the estimated population (18,000).

There were also reported 976 cases of tonsillitis, 569 of arteriosclerosis, 305 of chronic arthritis, and 87 of acute rheumatic fever.

The difference in the percentages shown in the two surveys is due in some measure to the more favorable age composition of the population of Essex County. We must also mention the fact that the physicians in Essex County were instructed to report cases of sickness among residents of the County only.

The Essex Survey also showed the etiology of the reported cases of heart disease. Of the 535 cases, 9 were given as syphilitic, 121 as rheumatic, and the rest, 405, as other types; in percentages—1.7, 22.6, and 75.7 respectively.

It is interesting to compare the number of cases of heart disease with all reportable communicable diseases. In the Rural Survey the cases of the latter totalled 3,212 as compared with 4,123 of heart disease; the corresponding numbers in the Essex Survey were 312 and 535. Cases of heart disease in both surveys exceeded the cases of reportable communicable diseases by practically one-third. These figures demonstrate in a striking manner the importance of the heart-disease problem in the State.

THE PREVALENCE OF HEART DISEASE

If we were to apply the imperfect and incomplete findings of the two surveys to the population of the entire State, we might say that cases of heart disease in the general population numbered between three and four hundred thousand. Another way to arrive at an estimate of the number of cases would be by means of the ratio of cases to deaths. During the fifty-two weeks of the Essex Survey the participating physicians filed 59 death certificates on which heart disease was given

*Sickness in Essex County. J. V. DePorte, New York State Journal of Medicine, November 1, 1929.

as the primary cause, or an average of 9 cases to one death. Time did not permit us to ascertain the number of deaths reported by the physicians in the Rural Survey. Assuming that the death rate was identical with that for the entire rural territory of the State, we find 295 as the estimated number of deaths, or an average of 14 cases to one death. Multiplying the number of deaths from heart disease recorded in the entire State in 1928 by these two ratios we get 310,000 and 480,000 cases respectively. Taking into consideration the various assumptions and estimates, we can say with a reasonable degree of certainty that there are at present in the entire State about three hundred thousand cases of heart disease.

A PLAN FOR THE STUDY OF HEART DISEASE IN THE STATE

The concluding paragraph of our report on the Essex County Survey read as follows:

> "The two surveys directed by the State Department of Health were made possible, through the *voluntary* cooperation of one hundred and twenty-seven busy practising physicians. This fact, in my opinion, is of transcending significance. The spirit of altruistic scientific inquiry manifested by these physicians encourages a hope of further investigations which could not be carried out in laboratories or offices of official and private health agencies."

A study of heart disease as seen in his everyday activities by the practising physician would seem to be a most appropriate continuation of the surveys conducted by the State Health Department. A group of diseases which incapacitates, partially or wholly, about three hundred thousand persons in the State is certainly a matter that cannot be excluded from the field of legitimate public health activities by the mere fiat of our individualistic tradition.

In the words of Dr. Theobald Smith,* "Public health operations deal with mass phenomena and consequently must use statistics . . . . As long as disease exists the operations of preventive medicine must be founded on our knowledge of disease . . . . All human inquiries are narrow and partial . . . . We must be satisfied with piecemeal work in the hope that occasionally some synthesizing genius will appear who can put the collected fragments together in some form acceptable to us and which will serve as a fresh pattern for further endeavors."

Dr. Smith speaks from the point of view of laboratory research, but his remarks apply equally well to statistical studies, such as we have in mind. I am confident that there will be no difficulty in securing the interest of a sufficient number of physicians who will agree to report periodically certain important facts relating to heart disease in their practice. The State Department of Health would collate the data and make them public from time to time. Unlike our earlier surveys,

*The Influence of Research in Bringing into Closer Relationship the Practice of Medicine and Public Health Activities. Am. J. Med. Sc., December, 1929.

this study need not be limited to any definite period and could be carried on continuously. Since the average number of cases of heart disease in a physician's practice is relatively small, it would be possible, without trespassing too much upon his time, to employ a fairly complete form which would give us certain facts about the etiology of each case, the age, sex, color, conjugal state, and other essential information relating to the patient.

I hope that the American Heart Association will coöperate with us in this undertaking, particularly during the formative period. The value of such a study is self-evident. It should enable us, among other things, to determine in the course of years the results of the various types of treatment employed and at the same time it would certainly be of definite educative value to the participating physicians themselves.

THE CARDIAC CLINICS OF NEW YORK; THEIR ORIGIN, AIMS AND ACCOMPLISHMENT*

EDWIN P. MAYNARD, M.D.
BROOKLYN, N. Y.

ONE of the great tragedies that has confronted all of us who have undertaken the care of hospital ward patients, has been the spectacle of the rapid breakdown of our adult cardiacs when they have left the wards and returned to their old environments. As we have seen them come back for readmission, sadly broken down, perhaps two or three weeks after we had restored them to compensation, we have been depressed by the gravity of the situation and the plight of these afflicted people who must somehow work to live.

Back in 1911 this picture of human suffering and economic waste stirred Dr. Hubert V. Guile and his associates at Bellevue Hospital in New York to form the first clinic or "class," as it was then called, for adult cardiac patients. The primary object was to provide after-care for cardiacs discharged from the hospital wards, to teach them their limitations, to guide them in industry, and in so doing, to prevent or postpone the next breakdown and subsequent hospital admission. The success of this pioneer effort is too well known to require description. Through the years it has served as a model and now under the leadership of Dr. John Wyckoff it stands as one of our ideal clinics.

By 1915 the growing importance of the problem of heart disease was beginning to be appreciated by a few discerning physicians and lay people, with the result that they formed themselves into the Association for the Prevention and Relief of Heart Disease. This organization began to attack the problem of heart disease from a much broader point of view than that of the clinic. Its objects were to gather information upon heart disease, to develop and apply measures to prevent heart disease, to seek and provide occupations suitable for patients with heart disease, to promote the establishment of special dispensary classes or clinics, to extend the opportunities for convalescent care and permanent institutional care, to encourage the establishment of associations with similar objects in other cities and to maintain a central office and clearing house.

The effort of the A.P.R.H. to promote the establishment of special dispensary classes for cardiac patients was very successful and soon twenty-six clinics were started. Through the efforts of the Executive Committee of the A.P.R.H. these clinics were organized into an association of cardiac clinics in 1917 under the chairmanship of Dr. Robert H.

*Read at the annual meeting of the American Heart Association, February 4, 1930.

Halsey, and in 1923 this association became the Committee on Cardiac Clinics of the Association for the Prevention and Relief of Heart Disease.

It would be tedious to follow the developmental history of this Committee on Cardiac Clinics up to the present time. Suffice it to say that this original Association of Cardiac Clinics is now the Committee on Cardiac Clinics of the New York Tuberculosis & Health Association.

This outline of the evolution of the Committee on Cardiac Clinics in New York City has been given to illustrate one way in which clinics in any city can be organized and can become part of a larger public health organization.

The trend of our times in business is toward mergers to overcome the waste of great numbers of smaller organizations all working for the same object. The same is or should be the trend in public health. The multiplicity of organizations in our large cities all striving for very similar purposes is appalling. Mergers in public health work offer a logical solution of the difficulty and the way the original Association of Cardiac Clinics has developed to its present position is a good illustration of how this unification of effort can be accomplished.

Now let us pass to the aims of an association of cardiac clinics and see how and in what measure they are accomplished. Because of lack of knowledge of associations in other cities, it will be necessary to speak of our own Committee on Cardiac Clinics of the New York Tuberculosis & Health Association. Perhaps the first and predominating aim was to maintain and improve the standard of work done in the member clinics. Before this could be undertaken it was necessary to determine just what those standards should be, both for minimum requirements and for an ideal clinic. This was done in 1923 and the result published in a leaflet for distribution to the member clinics. By this step the committee provided itself with a measure by which it could not only judge the work of its member clinics, but establish admission requirements for new clinics.

The Executive Committee of the Committee on Cardiac Clinics was then provided with a secretary whose main duty is to visit member clinics, explain the organization and its aims to them, assist them in every way possible and report back to the Committee the status of each member clinic. In most instances the way in which improvements are initiated is by the suggestion of the secretary to the social worker or clinic chief at the time of the visit. The clinics usually respond to the best of their ability. However, if they do not, the executive committee studies the situation with great care and then makes a written recommendation to the clinic chief, to the hospital superintendent or to both. If these measures fail and the standard of work in the clinic remains below the minimum requirements for membership in the Committee on Cardiac Clinics, the board of trustees of the hospital is ad-

vised of the situation. If they fail to act, the clinic is dropped from membership. Fortunately this drastic step has been necessary in only one instance during the life of the organization. Thus a great deal of the time of the secretary and of the Executive Committee is devoted to the study of actual conditions in clinics and of methods of improving them.

One of the great difficulties in coördination of any sort of medical work lies in the lack of uniformity in terminology. The descriptions of the same disease or set of phenomena vary greatly. A group of workers in one clinic may speak of the functional capacity of a patient for instance, using terms that would mean very little to a group in another clinic. Early in its history, the Association of Cardiac Clinics recognized this and in 1921 adopted a system of functional diagnosis and classification based upon functional capacity as gauged by the patient's ability to carry on ordinary physical activity. This classification has been modified only slightly since its adoption and is familiar to you all. It has been of inestimable value in giving us a common standard or measure by means of which we can convey ideas to each other about the functional capacity of our patients. Its use is rigidly required, especially in applying for admission to convalescent homes.

In the matter of records of cardiac patients, the Association soon discovered that there was no uniformity whatsoever. Each member clinic had its own method and the amount and quality of the data recorded depended upon the interest of the physician in the clinic. The records were in no sense comparable. By 1923 record forms for history, physical examination and follow-up notes had been devised, which after much deliberation were adopted by the Association. These forms are now in use in twenty-seven clinics.

Aside from their great value in improving the records in the clinics and for statistical research, they have developed into a useful instrument in the hands of the Executive Committee of the Committee on Cardiac Clinics. This has come about in an interesting way. The Research Committee of the Heart Committee issues these charts to the clinics that desire them. At first they are issued in single sheets for which the clinics pay a moderate charge. The statistical workers of the Research Committee check up the quality of these records and after they are satisfied of their accuracy, they recommend to the Research Committee that these charts be issued to the approved clinics in duplicate and free of charge. At stated times the duplicate pages are torn off and collected for study by the Research Committee. At the same time these approved clinics are offered the assistance of statisticians from the Research Committee who work in the clinics during clinic hours helping the physicians with the records.

Very close coöperation exists between the Research Committee and the Committee on Cardiac Clinics. As was stated before, the main

interest of the latter is to maintain and improve the quality of work done in the member clinics. Through the studies of the Research Committee, the Committee on Cardiac Clinics can readily learn the quality of the records and therefore of the work done in the clinics that use the uniform charts. Good records and good work go hand in hand. When the records from a given clinic show persistent inaccuracies it is of vital interest to the Committee on Cardiac Clinics to remedy the situation. This it undertakes, usually by personal interviews between one of its own members and the chief of the clinic concerned. The Committee had found this phase of its work one of great delicacy and feels that it should not be entrusted to a lay worker. When properly approached, the response of the physicians in the clinics has been, with few exceptions, satisfactory. When the uniform charts were first devised they were looked upon purely as devices for statistical research. Now we have come to realize that they are one of the best means by which the medical work in the clinics can be evaluated and improved.

In another matter of uniformity, the Committee on Cardiac Clinics is indebted to the Criteria Committee. After years of effort, originating in the old Association of Cardiac Clinics in 1923, a nomenclature for heart disease has been devised and with it a set of criteria for use in connection with the nomenclature. This material is now published in a small book by the American Heart Association. It marks a great step in the progress toward a common language in the Cardiac Clinics.

Our executive secretary had not been long in the field when it became apparent that the clinic chiefs and social workers had no idea of the number of patients in their clinics, how many were active in attendance and how many were lost. Furthermore, they had little knowledge of the efficacy of their social service follow-up. The clinic chief did not know whether his clinic was growing and healthy or declining and encumbered with deadwood. Furthermore, the Committee on Cardiac Clinics had no idea of the heart situation as a whole in the city as expressed by the volume of work done in all the clinics.

To remedy this situation, a system of clinic bookkeeping was introduced and also a monthly report form modelled after the one in use in the tuberculosis clinics. At first, great difficulty was encountered in getting the clinic bookkeeping in such shape as to yield the data for the report, but after much effort by statisticians and social workers, the system was inaugurated. By the mere use of this form, the social worker and the clinic chief now have an accurate picture of the state of their clinic. They know how many active cases they are carrying, how many were discharged and the reasons, how many died and in how many the follow-up was unsuccessful. Furthermore, the Committee on Cardiac Clinics tabulates these reports in such a way that a fairly complete picture of the work done in the clinics is available each year.

In 1929 the Committee on Cardiac Clinics with the help of Dr. Philip S. Platt made a statistical analysis of the figures for 1928. It was realized at the outset that since this was the first year that many of the clinics were reporting, there would be many unavoidable statistical

Committee on Cardiac Clinics of Heart Committee

New York Tuberculosis and Health Association

244 MADISON AVENUE, NEW YORK CITY

Report of Children's / Adults' Cardiac Clinic of ..... Hospital For Month of ..... 192..

	ADULTS (16 YEARS AND OVER)	CHILDREN (UNDER 16 YEARS)	TOTAL
I. Composition of Case Load:			
1. Old Cases Brought Forward			
2. New Admissions			
3. Readmissions			
4. TOTAL			
II. Disposition of Case Load:			
A. Closed (Discharged, Transferred or Dropped)			
5. Diagnosed Cases of Organic Heart Disease (Class I, II, III)			
6. Abnormal Signs and Symptoms (Class E)			
7. Potential Heart Disease (Class F)			
8. Non-Cardiac (Other than Classes E and F)			
9. TOTAL			
B. Open (Balance to be Brought Forward Next Month)			
10. Diagnosed Cases of Organic Heart Disease (Class I, II, III)			
11. Abnormal Signs and Symptoms (Class E)			
12. Potential Heart Disease (Class F)			
13. TOTAL (10 + 11 + 12; also 4 — 9)			

DISPOSITION OF CLOSED CASES (Item 9)

Discharged:	Transferred:	Dropped:
14. Non-Cardiac	19. To Other Clinic or Institution	24. Not Found
15. Maximum Benefit Secured	20. To Other Dept. of Hospital	25. Follow-Up Unsuccessful
16. Clinic Treatment not advised	21. To Private Physician	
17. Died	22. Referred for Opinion Only	
18. Total	23. Total	26. Total

27. TOTAL CLOSED (18+23+26) .....

COMPARATIVE CONDITION OF CLOSED CASES (Item 9)

28. Etiological Type:

Congenital	
Rheumatic	
Syphilitic	
Arterio-Sclerotic	
Hypertensive	
Unknown	
Other	
Multiple	
None (Non-Cardiac)	
Total	

29. Functional Capacity:

ON ADMISSION (A)		ON DISCHARGE (B)						
		Class						
Class	Total	I	II A	II B	III	E	F	Non-Cardiac*
I								
II A								
II B								
III								
E								
F								
Non-Cardiac*								
Total								

30. Clinic:

Number of Clinic Sessions during month	
Total Clinic Hours during month	
Total Clinic Visits during month	

31. Personnel:

	NUMBER ON STAFF	TOTAL HOURS OF SERVICE DURING MONTH
Physicians		
Nurses		
Social Workers		
Volunteers		
Clerks		

32. Social Service Visits:

Number Routine Follow-Up Visits	
Number Social Case Work Visits	
TOTAL	

33. Changes in Clinic Hours, Sessions or Staff:

Signed .....
(Medical Social Worker)

*Other than Classes E and F

FORM 7-2M-2-29

SEE REVERSE SIDE

Fig. 1.

inaccuracies in the compilation. Nevertheless it was thought worth while to make a beginning and eighteen graphic charts were drawn comparing the forty-two clinics in many important points.

Figure 1 shows the monthly report form which is filled in by the

social worker in each clinic every month. Using data derived from these reports the graphic analysis of various phases of the work in the clinics in 1928 were made.

Figure 2 is a study of the percentage of new admissions per year. The clinics had been reporting regularly only for a short time before

Clinic Key No.	Total Case Load	New Admissions No.	New Admissions %
1....	122	87	71.3
2....	260	185	71.2
3....	188	128	68.1
4....	278	161	57.9
5....	110	63	57.3
6....	312	146	56.8
7....	331	170	51.4
8....	679	320	47.1
9....	238	112	47.1
10....	456	215	47.1
11....	219	103	47.0
12....	73	31	42.5
13....	90	38	42.2
14....	144	60	41.7
15....	153	63	41.2
16....	144	59	41.0
17....	254	95	37.4
18....	224	83	37.1
19....	380	138	36.3
20....	557	191	34.3
21....	367	126	34.3
22....	265	89	33.6
23....	473	157	33.2
24....	458	146	31.9
25....	161	51	31.7
26....	640	183	28.6
27....	215	61	28.4
28....	184	51	27.7
29....	184	48	26.1
30....	81	21	25.9
31....	196	50	25.5
32....	191	44	23.0
33....	382	88	23.0
34....	365	80	21.9
35....	108	23	21.3
36....	211	44	20.9
37....	527	109	20.7
38....	413	82	19.9
39....	305	58	19.0
40....	253	45	17.8
41....	492	85	17.3
42....	150	21	14.0

Fig. 2.

1928, and because of this, many statistical inaccuracies are undoubtedly present in the first year's work. For this reason the clinics have been listed by arbitrary numbers. A study of Figure 2 shows that there is a great variation in the growth of the individual clinics. Some are new and therefore show a high percentage of new cases and others do not appear to be growing at all.

In order to learn what was the average clinic practice and to throw some light on the efficiency of follow-up work in the clinics, similar tables were constructed to show the number of clinic visits per patient per year and the number of patients dropped per year. It does not necessarily follow that clinics with the fewest patients dropped had the best record. Perhaps in those clinics the standards of attendance and coöperation were too low and cases were kept enrolled which should have been closed.

It cannot be emphasized too strongly that the graphic charts of 1928 were an experiment to try out the method. It is planned to make a similar study with the more accurate figures of 1929, in the hope that in the future some sort of statistical measuring rod can be devised to demonstrate what constitutes good clinic practice as regards the matters studied in the charts.

There is one more phase of the work of the Committee on Cardiac Clinics that should be mentioned under the heading, educational. The committee holds two scientific meetings a year to which all the physicians and social workers in the clinics are invited. The program of one of these meetings is devoted entirely to the presentation of papers based upon work done in the Cardiac Clinics and to discussion of clinic problems. The other is of wider scope and speakers are usually invited from other cities to bring a fresh point of view to our clinics. The educational idea is carried still farther in that courses are arranged from time to time for social workers.

Enough has been said to make it obvious that the work of the Executive Committee of the Committee on Cardiac Clinics is both varied and interesting. In the constant effort to keep up and improve the standards in our clinics many difficult and delicate problems arise which require patience and tactful handling. In some instances, the Committee has failed, but more often, thanks to the intelligent coöperation of our clinic physicians, it has succeeded. Its objectives are twofold, to provide better care for the individual patient, and to secure accurate data for statistical research. As we grow older in the work we come to realize more and more clearly that careful records in themselves result in better care for the patient.

Department of Clinical Reports

COMPLETE HEART-BLOCK OF SEVEN YEARS' DURATION IN A CHILD RESULTING FROM INJURY

T. Homer Coffen, M.D.
Portland, Ore.

HEART-BLOCK (auriculo-ventricular dissociation) in children is not common. In 1922 Rosenson[1] collected 36 cases of heart-block in infancy and childhood and reported an additional case in a girl of 11 years with congenital heart disease. In the collected cases etiological factors were: diphtheria, 16 cases; rheumatism 2; congenital syphilis 1; cardiac tumor 1; nose and throat infection 1; associated congenital heart lesion 12; unknown 3. Heart-block occurring during diphtheria presents the gravest outlook, the mortality being 93.7 per cent in the collected cases.

Ernst Lorenz[2] in 1927 reported a case of functional partial heart-block. Romberg and White,[3] McIntosh[4] and Le Conte[5] reported heart-block in young children, probably of congenital origin and Sands[6] reported a case of a child of 6 years with three distinct attacks, one after scarlatina, one after diarrhea, with recovery after a few days. After nine months the block was still present.

Rosenson[1] reported the case of a boy of 10½ years who had heart-block resulting from a blow on the precordium. At this time Rosenson said this was the only case thus far reported with this etiology.

Morris Kahn and Samuel Kahn[7] in an admirable review of cardiovascular lesions following injury to the chest point out the variety of changes in rhythm and function resulting from injury from blows on the chest. "The superficial position of the heart and precordium exposes them to danger from injuries to the anterior chest wall. The character of the effects depends upon the status of the heart cycle at the moment of the accident, the resilience and resistance of the chest wall and the force, momentum and direction of the injuring force." They report auricular fibrillation, injury to valves, precordium, or rupture of the heart from chest injuries.

REPORT OF CASE

Kenneth K., now 10 years, fell from a wood pile four or five feet high when 3 years old (1922). The mother says that in trying to catch himself he hurt or fell

on the chest. He came into the house crying and complaining of pain in the upper abdomen. The mother did not see the fall; no bruises or wounds were seen. There was no cyanosis nor dyspnea. The child was in pain all night. The mother found the pulse rate 65 in the morning and took the child to the nearest town (18 miles distant), where it was found that the pulse rate was then 50. A diagnosis of intestinal obstruction was made and the child operated upon, but no obstruction was found. His pulse rate dropped to 36 and his condition was critical for a few days. In the next three months there was gradual improvement and he has been well since. His mother has been engaged in child welfare work and is certain that his pulse had always been normal. The child never had scarlet fever, diphtheria or other illness. Just before the accident he scored 98 per cent in a baby clinic examination.

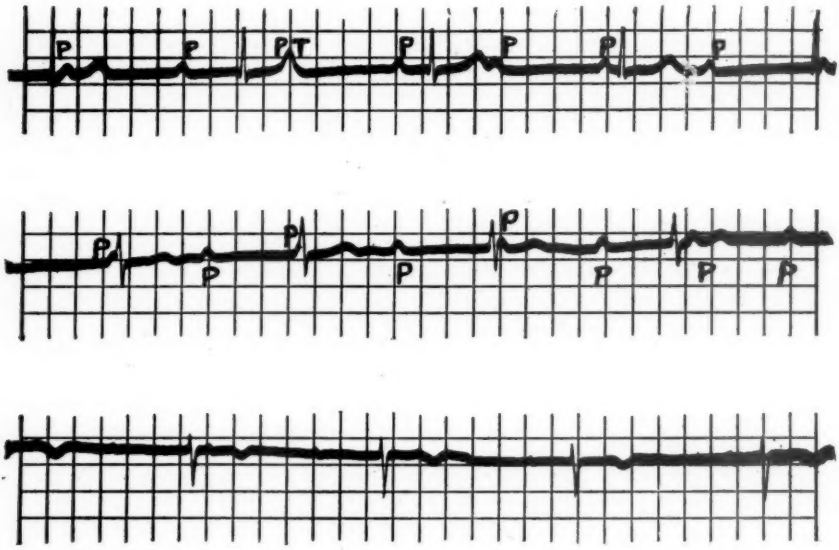


Fig. 1.

Examination: The boy was healthy in appearance, height 4′ 7¾″, weight 70½ lbs., color good, no cyanosis, pallor or abnormal pulsations in precordium or superficial vessels. The pulse was 48, regular. Eyes, ears, nose and throat negative. There was no adenopathy. The thyroid was not enlarged. The chest was symmetrical and negative. The heart borders were within normal limits though there was some increase in dulness in the waist. There was a systolic murmur at the pulmonic area, louder in the recumbent position. There was also a faint systolic murmur at the apex. In the recumbent position there was a positive wave suggesting auricular contractions about 80 per minute while the pulse rate was 48 per minute. The blood pressure was 90-95/80. The abdomen was negative, the liver not felt. There was no clubbing of fingers. The reflexes were normal. Urine and blood examinations were normal. Electrocardiograms show complete auriculo-ventricular dissociation, the auricular rate being about 80 while the ventricular rate is about

50. The P-complexes show no time relation to RT. The latter are normal (see Fig. 1). An orthodiagram (see Fig. 2) shows the globular type of heart with dulness in the waist (region of left auricle). In the right anterior oblique view the retrocardiac space was narrow but no abnormal notching of the esophagus was noted when barium was swallowed. In the left anterior oblique the left ventricle overrode the spine by about ½ inch. There were no abnormalities about the aorta or pulmonic area. There was no enlargement of the thymus or thyroid.

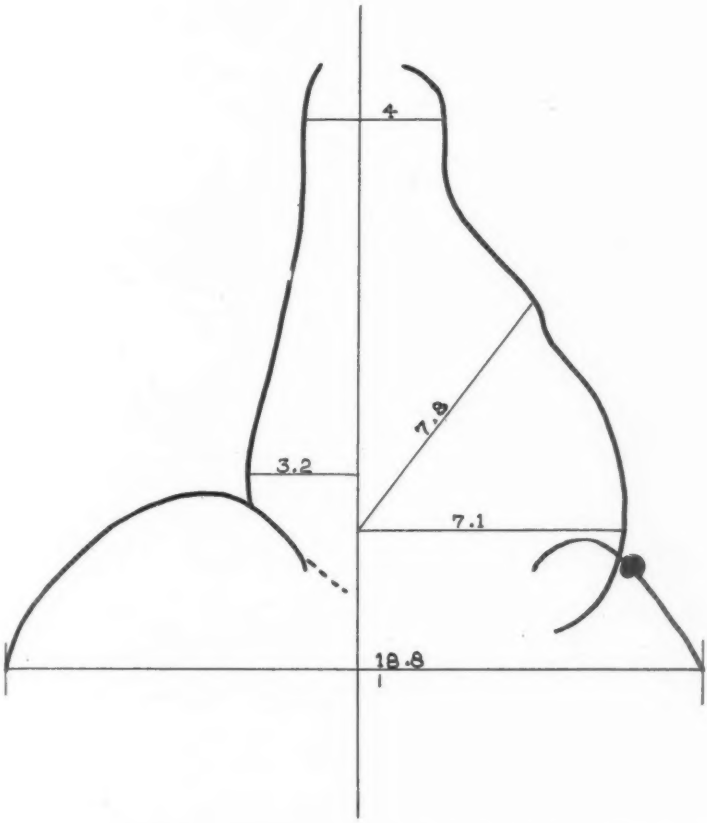


Fig. 2.—Kenneth K. April 20, 1929. Lungs: Entirely negative. Heart: Globular type but with very full waist. In the 1st oblique the space is narrow but no abnormal notching noted with barium. In the 2nd oblique the left ventricle overrides the spine ½ inch and on deep inspiration just failed to clear. The aortic window is clear.—E. L. B.

SUMMARY

A case of complete auriculo-ventricular dissociation is presented, occurring in a healthy appearing and physically active boy 10 years of age. It is presumed that an injury, sustained seven years before produced this condition, for he was a healthy child at the time of the accident. The pulse rate was very slow afterward and has remained so.

REFERENCES

1. Rosenson, Wm.: Am. J. Dis. Child., **28**, 594, 1924.
2. Lorenz, Ernst: Ztschr. f. Kinderheilk., **43**, 552, 1927.
3. Romberg, E. C., and White, P. D.: Boston M. & S. J., **190**, 591, 1924.
4. McIntosh, Rustin: Am. J. Dis. Child., **34**: 965, 1927.
5. Le Conte, M.: Bull. d. l. Soc. Méd. d. Hôp. Paris, **48**, 488, 1924.
6. Sands, M. J.: Arch. Pediat., **50**, 343, 1923.
7. Kahn, Morris H. and Kahn, Samuel: Ann. Int. Med. **2**, 1013, 1929.

INSULIN SHOCK AS THE CAUSE OF CARDIAC PAIN*

Case Report

Kenneth B. Turner, M.D.
New York, N. Y.

THE symptoms of hypoglycemic shock respond promptly to the administration of glucose and usually cause little concern on a diabetic service. In fact, many clinicians believe that all diabetic patients who require insulin should be made to have at least one "shock" before leaving the hospital in order that they may become familiar with the premonitory symptoms of this condition. That this practice is not free from danger, particularly in patients with arteriosclerosis, is suggested by the following case report:

The patient was a fifty-eight-year-old Italian housewife. She was first seen in the Metabolism Clinic in February, 1923, when she stated that she had had diabetes for four years. She was advised as to her diet and did well until January, 1925, when an infection lowered her tolerance for sugar so that she was forced to enter the hospital for dietary regulation. She remained in the hospital for two weeks, became sugar-free, and was discharged without insulin. At the time of this first admission, cardiac symptoms and signs were not impressive. She stated that for several years she had had slight dyspnea and palpitation on exertion and also an "occasional feeling of distress over the left chest." The history is not exact, but it is clear that the patient was not greatly disturbed by these symptoms. On physical examination the heart did not seem enlarged, the sounds were of good quality and regular and there were no murmurs. The blood pressure was 155/90 mm. The radial arteries were firm. There were no electrocardiograms or x-ray studies at this time.

After discharge she remained under observation in the Metabolism Clinic until June, 1928, when it was decided that she should have insulin, and she was readmitted to the hospital. At that time she denied any cardiac symptoms. An x-ray film of the heart showed considerable enlargement, especially to the left. Calcification was apparent in the aortic arch. Her blood pressure was 135/85 mm. The electrocardiogram was essentially negative. The blood Wassermann was negative. She was not anemic.

On June 8, during the evening, she had an insulin shock. She became pale, covered with perspiration, nervous, and had a marked tachycardia. During this, she had an attack of severe precordial and substernal pain that radiated down the inner border of the left arm and into the left little finger. This pain was relieved promptly by amyl nitrite. A similar attack, under similar circumstances, occurred on June 22, and another on June 26. She was discharged again to the Metabolism Clinic and followed there. She had no more insulin shocks and no more anginal pain.

*From the Department of Medicine, College of Physicians and Surgeons of Columbia University, and the Presbyterian Hospital.

In this elderly woman with an enlarged heart, it is interesting that the first insulin shock with its attendant tachycardia promptly produced a severe seizure of cardiac pain that twice recurred under similar circumstances. This clearly indicates the necessity for proceeding cautiously with the administration of insulin in diabetics who may have coronary sclerosis, lest unfortunate consequences result from insulin shock.

Department of Reviews and Abstracts

Selected Abstracts

Rösler, Hugo: On Congenital Isolated Dextrocardia. Wien. Arch. f. inn. Med. **19:** 505, 1930.

This paper contains a very extensive and complete discussion on the subject based upon a thorough search of the literature from which the author has collected 38 cases verified by autopsy and 126 cases not so verified, but the author stresses the importance of anatomical verification, because otherwise partial situs inversus cannot be excluded.

The author describes his own seven cases. In the first case there was an isolated inversion of the ventricles and the large trunks as well as a transposition of the aorta alone into the right ventricle. The second case was remarkably free from symptoms, the third had peripheral malformations. On x-ray three cases had high dextroposition, twice there was a mirror-position of the heart, five times atypical shape of the cardiac shadow. In five cases the left diaphragm was higher than the right. One electrocardiogram was normal, one was diverted, one completely atypical, two had negative T-waves.

The 38 cases verified by autopsy are described and interpreted. The average age was eleven years, and various cardiac malformations were frequent and there were cases with situs solitus (the heart alone being transposed) combined with normal or transposed origin of the great vessels. The isolated congenital dextrocardia is not a malformation sui generis. No case is known with transposition of all parts of the heart and no case is known in which the heart and the great vessels were not malformed.

In the 126 cases described clinically the average age was 23½ years. Great exercise tolerance was noted in many of these cases. In many there were other malformations, especially defects in the muscles to the ribs. The apex beat was well demarcated, diffuse or even impalpable. Seventy-five cases had x-ray photo graphs, some had mirror pictures, others had atypical shadows. In the mirror forms the high dextroposition of the aorta predominates, in the atypical shadow forms the normal leftsided position. In most cases the right diaphragm was low, this difference between the diaphragms being caused by the cardiac position and not by the liver.

Twenty-nine electrocardiograms were obtained, 8 were normal, others had negative T-waves, with or without axis deviation. In these cases T-wave abnormalities had no prognostic significance. Q-waves were frequent, probably due to malposition. Disturbances of rhythm were rare. When the x-ray showed mirror picture, the electrocardiogram was usually inverted, the atypical x-rays (not "mirrored") would show positive electrocardiograms. The electrocardiographic interpretation was difficult on account of lack of material for verification, the anatomical relations being so very complicated and even the conduction system might be abnormal.

In a few cases it is possible for the patients to obtain a high age without impairment of cardiac function, but as a rule the prognosis is gloomy. About 350 references to the literature are appended.

Baker, Benjamin M.: The Effect of Cardiac Rate and the Inhalation of Oxygen on Transient Bundle-Branch Block. Arch. Int. Med. **45:** 814, 1930.

It is the purpose of this communication to record further observations on temporary bundle-branch block and to discuss the nature of transient disturbances in the conduction of the excitatory process within the ventricular specialized tissue. A case report is made of a patient who showed signs of transient abnormal ventricular conduction whenever the period of rest between ventricular systoles was of sufficient length. The electrocardiograms showed that the intraventricular conduction time was within normal limits and whenever this period was somewhat shortened, faulty conduction ensued. Rest in bed and the administration of appropriate doses of digitalis afforded means of slowing the ventricular rate. It was also noted that the aberrant complexes disappeared during inhalation of oxygen. Also during the administration of oxygen although the cardiac rate was increased by exercise well above the point provoking delayed conduction in all previous observations the normal appearance of the ventricular complexes was retained.

Dressler, Wilhelm: On the Formation of Interference, Dissociation and of Retrograde Propagation of Ventricular Extrasystoles. Wien. Arch. f. inn. Med. **19:** 611, 1930.

The authors have studied cases where a disturbance of impulse formation has led to a permanent nodal rhythm.

Report follows of a case with nodal rhythm, where for some reason the complexes discontinue to travel backward to the auricle. Then impulses originate in the sinus node and with delayed conduction time travel to the ventricle where they produce complexes which have a tendency to become atypical. This change from nodal to auricular rhythm occurred abruptly. Changes in shapes of P-waves suggest various places of origin for auricular contractions; such interchange of various centres of origin is not uncommon where the superior centre is depressed, especially as a vagus effect. The patient also showed ventricular extrasystoles which were followed by a wave strongly suggesting a P-wave—probably these are retrograde extrasystoles. These must be considered a rare occurrence.

Another patient had sinus depression, lasting as long as 3.5 seconds if he held his breath; during this period escaped beats or nodal rhythm would occur, this would occasionally be interrupted by complexes which the authors thought originated high in the A-V node. Some years later this gave place to a definite nodal rhythm.

These observations led the authors to this problem: Why, in some cases, does nodal rhythm fail to produce auricular contractions? Authors produce evidence that the nodal impulses do not even travel back toward the auricle, the retrograde conductivity being so difficult as to prevent some of the nodal impulses from traveling backward. In such cases where nodal rhythm is regularly associated with auricular contractions, the conduction system has been trained to retrograde conduction; this is supported by the frequent occurrence of auricular complexes after extrasystoles occurring in nodal rhythm.

Vagus effect might cause the rhythm to change from nodal to "interferenzdissoziation," and sympathetic stimulation would cause the reverse change.

Dressler, Wilhelm: Disturbed Conduction in the Auricle. Med. Klin. **25:** 185, 1929.

Lewis wrote in 1925: "So far as we know, 'aberration' is peculiar to the ventricle, the auricle is exempt, because in its structure it possesses no special conduction paths."

Scherf and Shookhoff produced nodal rhythm in dogs; when the nodal rhythm was resumed after extrasystoles, the nodal extrasystoles were associated with changes in P-waves. These might even become positive. This is explained by the impulses using different pathways through the auricle. Rothberger and Scherf ligated the upper and lower end of the sinus node and obtained a sinus rhythm with negative P-waves. They thought this was due to atypical conduction paths, for ligation along the sides of the sinus node produced no change in P-waves. Therefore, P-waves of atypical auricular activation may be positive and of sinus rhythm may be negative, all depending upon the journey of the impulse through the auricle.

Two case reports follow: One patient had auricular extrasystoles preceded by an upright but atypical P-wave and shortened P-R interval. This shortened P-R interval causes the author to diagnose nodal extrasystole originating high in the node and with positive P-wave, and on the basis of the experimental evidence quoted above, ascribes the form of the P-wave to the atypical retrograde course of the impulse, finding the usual path refractory. He emphasizes the rarity of such a finding. As a rule in nodal rhythm P is positive when it recedes the R, negative when it follows. The author argues against the interpretation of wandering pacemaker in cases of nodal rhythm where the P-R interval changes as well as the shape of the P-wave. A critical discussion on other reported cases follows.

Another patient had partial block, some auricular impulses being entirely blocked, the P-waves following such pauses all had atypical shapes. The atypical P-waves had presumably the same origin as the regular ones, the timing being absolutely regular. Therefore the atypical shape must be due to an atypical course. To the author's knowledge this is the only case on record where P-waves changed shape under these circumstances. Moenckeberg has shown that pathological processes in the auricular myocardium may cause partial or complete heart-block. This may be the explanation in this second case.

Dressler, Wilhelm: Dissociation and Interference in Partial Heart-Block. Ztschr. f. klin. Med. **111:** 23, 1929.

In pararhythmias two centers of unequal frequency are active, the slower being protected by blocking from the other. Occasionally equal frequency of both centers may lead to a dissociated function of auricle and ventricle; this state in the course of change of one rhythm to another has wrongly been termed wandering of the pacemaker in the A-V node. Author reports a case with two pacemakers of equal frequency, and on the basis thereof discusses the conditions under which both centers become active.

A case of bradycardia (44 beats per minute) showed an electrocardiogram with ventricular complexes at even intervals, while the interval between the P-waves was constantly changing. The change in auricular intervals did not influence the P-R intervals. There the authors were not dealing with partial heart-block, but with automatic ventricular rhythm, the impulses of which did not affect the auricular rhythm, because they found the auricles refractory from the preceding auricular contractions. This case makes the author ask: Under which conditions will two centers of equal frequency produce a dissociation of two cardiac sections? That can only be done when the two centers beat quite or almost synchronously. If impulses originate in one focus too early the impulses will cause a contraction of the entire heart, preventing further ectopic impulse formation.

Another case showed complete heart-block with double rhythm, two auriculo-ventricular to one automatic ventricular contraction. Sometimes an auricular systole occurred, when a refractory period of the ventricle had passed off and before the

next ventricular contraction; in this case the impulse was conducted through and resulted in a ventricular contraction.

The two factors which determine the disturbance of rhythm are frequency of the impulses and the duration of refractory period. But the refractory period varies from beat to beat, but in all cases does it exceed the sinus-interval, for otherwise sinus rhythm would be established; it is shorter than twice the sinus-interval, for if it approaches or exceeds, the auricle again determines the cardiac contractions (2:1 heart-block), or the same condition occurs as in case 1.

Extrasystoles would prevent transmission of auricular impulses, which according to the time of their occurrence ought to have been transmitted, thus showing retrograde transmission of the impulses, sufficient to cause refractory blocking of the conduction path.

These cases make intelligible the complete A-V dissociation in ventricular tachycardia, because the ventricular intervals are shorter than the refractory period of the conduction system plus the conduction time of another impulse.

Another case showed a marked arrhythmia of the auricles, varying from .59 to .95 seconds. The ventricular complexes also had irregular intervals and fell into two groups, in one the interventricular intervals changed between .98 and .105, in the other between 1.23 and 1.26; long and short periods alternated, every other complex was automatic, and every other one (the ones following the shorter periods) was conducted down from above. So here was a 2:1 block interfering with a regular ventricular rhythm.

Such cases as these are rare in the literature. They are characterized by a combination of sinus rhythm and automatic ventricular action, the impulses of which are not conducted backwards. The refractory phase of the bundle is longer than at least one sinus interval, so that the automatic ventricular period is shorter than the interval between two effectively conducted auricular impulses. Only such auricular impulses are conducted to the ventricle as fall after the refractory period of the bundle, and disturb here, being premature, the regular ventricular rhythm. But if the automatic ventricular interval is shorter than refractory period plus conduction time, then complete dissociation occurs.

These arrhythmias are not caused by disturbances in conduction, but primarily by the occurrence of a second focus of impulse formation. This may be the cause of complete block more frequently than is generally supposed. Both from the point of view of prognosis and of treatment it is important to distinguish between complete heart-block and partial block with dissociation; the latter having a more favorable prognosis. Also a complete block with certain time relation may mimic a 2:1 block.

The authors finally give the following classification of these "pararhythmias"; that is arrhythmias with two cardiac foci of impulses.

1. Interferences: one focus dominates while another focus (the interfering one) manifests itself in the entire heart or a part thereof.
2. Total dissociation of auricle and ventricle.
   a With maintained conduction system.
   b With complete block.
3. Dissociation with interference is a combination of 1 and of 2*a*.

Fischer, Robert, and Kiss, Aristid: A Contribution to the Knowledge of Pararhythmia. Deutsches Arch. f. klin. Med. **164:** 73, 1929.

The pararhythmia was observed in a patient with rheumatic heart disease, and with a P-R interval of 0.260 sec. to 0.280 sec. and automatic ventricular contractions, singular or in groups, which sometimes dominated the rhythm. The shape of these extrasystoles was definitely atypical. When sinus rhythm was established after a period of such extrasystoles, the P-R time was at first very must pro-

longed and became gradually shortened down to 0.280 sec. This is the opposite of what is seen in certain forms of heart-block (Wenckebach's periods). This variation in conduction time actually determined the onset and cessation of idioventricular rhythm. On account of the disturbance of conduction this was not considered dissociation with interference, in which never more than one normal beat at a time interrupts the idioventricular rhythm, which is of higher frequency. It is only because the A-V conduction time is constantly shortened that sinus beats continue to come through, though the sinus rhythm is shorter than the idioventricular rhythm. Probably this disturbance of conduction is also responsible for the failure of retroconduction of the extrasystoles. While the pararhythmia existed it was occasionally interfered with, because the P-P intervals were but very slightly longer than the P-R intervals.

Dressler, Wilhelm: Permanent Nodal Rhythm, With Attacks of Unconsciousness Caused by Ventricular Flutter. Klin. Wchnschr. **8:** 165, 1929.

Case report. Male fifty-three years of age, complained of dyspnea and dizziness and lately also of attacks of unconsciousness, which were becoming more frequent. Nodal rhythm was present at a rate of 38 per minute. Probably the focus was low in the node. Sinus rhythm was occasionally present.

The attacks of unconsciousness were ushered in by a feeling of flutter over the precordium and they lasted two to three minutes. He also occasionally had attacks of "pulling" in the chest associated with dizziness. These light attacks were observed electrocardiographically: at first single and later massed heterogeneous extrasystoles occurred at a rate of 200 per minute; in certain series they were homogeneous and reminded the authors so much of auricular flutter, that they called them ventricular flutter. During these attacks the pulse was not felt.

The author believes coronary disease to be the etiological factor. The differential diagnosis is from Adams-Stokes' syndrome, particularly because both forms of attacks occur with heart-block, and is considered relatively important; the prognosis is more serious in flutter, and the drugs recommended in the treatment of Adams-Stokes' syndrome (strophanthin, adrenalin, barium chloride) are apt to aggravate flutter.

Geraudel, E.: The Sign of "Satellite Auricular Contraction" in Adams-Stokes' Syndrome. Arch. d. mal. du coeur. **23:** 18, 1930.

As a result of previous work the author begins with the following conclusions:

The right auricle anatomically consists of two parts, one originating from the sinus and one from the atrium. The sinus vestibule represents the most upper part of the cardiac tube. It is connected on one hand to the atrial cavity and on the other to the ventricular cavity.

Strictly speaking the ventricular cavities communicate with the sinus cavity not with the atrial cavity. This sino-ventricular communication consists of two parts, a lower corresponding to the bundle and a higher one. Therefore, Adams-Stokes' syndrome may occur due to interference with conduction high in the vestibule or lower in the bundle.

The author has also shown that electrocardiographically the negative P-wave is of special importance. This has hitherto been considered evidence of auricular contraction occurring from below upward (retrograde contraction), while the positive P-wave showed the contraction spreading from above downward. The author provisionally assumes that the ectopic auricular focus is placed in the subeustachian sinus of Keith. The sinus communicates with auricular and ventricular cavities through four muscle bundles: (1) one to the superior auricle, (2) one to the inferior auricle, (3) a second one to the inferior auricle, (4) one with the ven-

tricles. That is; the lower auricle is controlled by two bundles which later unite in a common trunk, one of which runs in the upper and one in the lower part of the auricle.

The normal auricular contraction is a combination of the activities of the upper and the lower centers, the latter being subordinate. Of the two bundles going from the lower center, one is the lower bundle to the lower auricle, another is the one to the ventricles. The evidence of activity of the lower center is included in the QRST complex, occurring during the iso-electric period between the S and T, and may be ignored.

If the waves from both auricular centers are superimposed, they may be difficult to differentiate.

The fact that the lower auricle is supplied both from an upper and a lower branch has important consequences. In the normal heart the impulse comes through the upper path and the contraction wave coincides with the upper auricle. Pathologically blocks may occur in various places, for instance, a partial block may occur in the lower bundle above the point where the bundle to the lower auricular center separates from the ventricular bundle. Then the lower auricle is controlled by the upper center; under certain circumstances, however, the partially blocked impulse traveling the lower path may find the lower center at a time when the refractory period following the upper impulse has passed off. Then results a lower auricular contraction coinciding with the ventricular contraction.

This coincidence of contraction of lower auricle and ventricle may also occur if the upper path to the lower auricle has been interfered with, so the lower auricular center is not rendered refractory by the impulse traveling the upper path.

The form of the P-wave has therefore a certain diagnostic value, determining the site and the extent of auricular block. One can determine whether Adams-Stokes' syndrome is due to block in the bundle of His or in the auricular pathways.

In view of this knowledge the author has reviewed 397 electrocardiograms from 49 patients with Adams-Stokes' syndrome.

Eleven had marked P-waves from the lower auricular center, in these therefore, the block is situated not in the bundle, but above the bundle. Records of these cases are given.

Henderson, Yandell, and Mobitz, Woldemar: The Constant Rate of Absorption of Ethyl Iodide Vapor and Its Significance as a Basis for Measuring the Circulation. Am. J. Physiol. **92**: 707, 1930.

This article represents a reply to the criticisms that have been raised to the use of this method of estimating the blood flow. The authors deal particularly with: (1) Analysis by means of iodine pentoxide, (2) The automatic sampling of alveolar air, (3) The coefficient or effective coefficient determining the passage of ethyl iodide into the alveolar air into the lungs.

The method of analysis by means of iodine pentoxide when properly used is reliable and accurate as well as simpler and more rapid than other methods. All methods of analysis for calculating the circulation should be controlled by estimations of the dead space.

Automatic sampling of alveolar air is now established as a reliable technic.

During quiet breathing no considerable error is involved in using as true alveolar air the last portion of full normal expiration.

The authors then discuss the various factors to be considered in determining the passage of ethyl iodide from the alveolar air into the blood in the lungs. They believe that the method is satisfactory.

Ernstene, A. Carlton, and Blumgart, Herrman L.: Orthopnea. Its Relation to the Increased Venous Pressure of Myocardial Failure. Arch. Int. Med. 45: 593, 1930.

The authors believe that the orthopneic position benefits the patient with congestive circulatory failure because it secures a maximum blood flow about the respiratory center and thereby relieves the patient from the distress due to partial asphyxia in that area. Accordingly, a patient with myocardial failure and increased venous pressure always tends to maintain an elevation in bed sufficient to keep the respiratory center above the meniscus of the column of venous blood extending upward from the right auricle. In the upright position, pressure in the veins about the respiratory center is kept more nearly normal than in any other position and the blood flow in the capillaries feeding these veins is increased to the maximal limit set by the existing myocardial failure.

In order to test the validity of the hypothesis, 82 comparisons of the height of venous pressure and the degree of orthopnea were made in 22 patients with uncomplicated myocardial failure of the congestive type. A parallelism between the two measurements was observed. In general, it was found that the higher the venous pressure the greater was the orthopnea.

When orthopneic patients were placed in the recumbent position with the head flat, simple elevation of the head by flexion of it on the thorax produced, almost without exception, conspicuous diminution of respiratory distress. This procedure favors diminution of the cerebral venous pressure but has no significant effect on the vital capacity of the lungs.

Jolliffe, Norman: Liver Function in Congestive Heart Failure. J. Clin. Investigation. 8: 419, 1930.

The frequency of clinical jaundice in a series of 231 patients with congestive heart failure was observed to be 2.1 per cent. Fifteen of the sixteen patients had some alteration in liver function though no characteristic type was found.

No parallelism between the degree of heart failure and the impairment of liver function could be noted in individual cases. There was perhaps a parallelism between the changes in liver function and the degree of edema and size of the liver.

The author believes that liver dysfunction induced by an attack of chronic passive congestion is not permanent and that when it persists following recovery from passive congestion an independent liver impairment should be suspected.

Dock, W., and Tainter, M. L.: The Circulatory Changes After Full Therapeutic Doses of Digitalis, With a Critical Discussion of Views on Cardiac Output. J. Clin. Investigation. 8: 467, 1930.

A critical review is made of the older and current views of the actions of digitalis on the circulation and an attempt has been made by experiments on dogs to correlate its influence on the heart and peripheral vessels with diminution in cardiac output. The action of the drug in man varies with the functional state of the circulation in determining the output. When the normal heart is studied in animals and probably in man, there is a reduction following the use of digitalis but in such pathological states as heart failure with chronic passive congestion, digitalis would seem to tend to increase the cardiac output and to restore the venous pressure to a normal level.

Smith, W. Carter, Walker, George L., and Hait, Howard L.: The Cardiac Output in Heart Disease. Arch. Int. Med. 45: 706, 1930.

In this study the authors have observed the response of the circulation particularly as regards the output of the heart in several types of heart disease in-

cluding complete heart-block, auricular fibrillation before and after the restoration of normal rhythm, subacute rheumatic fever and chronic rheumatic valvular disease. None of these cases showed signs of congestive failure of the heart. The output of the heart was measured by the method of Field, Bock, Gildea and Lathrop.

The output of the heart was studied in three patients with complete block. The minute output of the heart was within the range of normal but each had a greatly increased stroke volume. Of the three patients with auricular fibrillation, the two with mitral stenosis showed an increase in cardiac output of one-fourth or more when the rhythm became regular. The cardiac output of one of these patients was the same with the irregularity of auricular fibrillation as with a regular sino-auricular rhythm caused by auricular premature contractions. When a regular rhythm without premature contractions was established there was a 29 per cent increase in the cardiac output. A third patient who had a pulse deficit of from 15 to 20 a minute but no mitral stenosis showed a corresponding increase in output when the pulse deficit was eliminated with digitalis. The patient with subacute rheumatic fever and with chronic rheumatic valvular disease associated with regular rhythm showed considerable fluctuation but was within normal limits.

With the onset of congestive failure of the heart there may be an increase in the output of the heart.

Deutsch, Felix: Variation in Heart Size, Especially the Diminishing Heart, Immediately After Exercise in Sports. Arbeitsphysiologie, **2:** 215, 1929.

The changes which the heart undergoes after exercise are not yet conclusively investigated.

Author studied participants in the Olympic games.

There is evidence that sportsmen's hearts are larger during the season when they are in training than during the "dead season." In one case the cardiac diameter was increased 5.1 cm. above the calculated normal. After the intense exercise of the fight, the heart was barely larger than the calculated normal; that is, it diminished during the exercise. Within a few days it regained its former size.

The greatest diminution amounted to 3.2 cm.; that is, one-fifth of the original size. All except four hearts diminished in size.

The author discusses the causes of this change: the tachycardia, the diminished amount of circulating blood (much blood remaining in the periphery, and much fluid being lost during exercise) and others are considered.

The diminution of size lasts until recovery occurs.

Kahn, Morris H.: Auricular Flutter Following Direct Injury to the Chest. Am. J. M. Sc. **179:** 605, 1930.

A case is reported in which following direct violence to the chest auricular flutter was discovered with signs of heart failure. The electrocardiographic records confirm the diagnosis. The patient died four months after the accident without post-mortem examination. The condition persisted in spite of treatment and was unimproved when last seen. The possibility of subepicardial ecchymosis in the auricular muscle is to be considered as the cause of the condition. It was noted in the electrocardiogram that the alternate ventricular cycles occur after every four and every two auricular contractions. In taking blood pressure measurements this produces an arrhythmia with the impression of a distinct pulsus alternans with a difference of 30 mm. in the systolic pressure between alternate cycles. This difference is equivalent to half the pulse pressure. The author surmises from this that the auricular function in this case was a most important one in producing

ventricular filling. Apparently every contraction of the auricle during flutter contributes its definite measure of blood to the ventricular volume.

It was also noted that preceding every ventricular cycle that follows two auricular contractions there is a normal P-R interval of 0.2 second. When four auricular waves occur, conduction becomes reduced to 0.14 second. It is thus seen that there is an improvement of conductivity following the longer periods of block.

Fordyce, A. Dingwall: Undetected Syphilis and Rheumatic Infection in Childhood. Brit. M. J. **1:** 530, 1930.

This study describes the details of nine children with rheumatic heart disease who were also congenital syphilitics and in whom the striking feature was the absence of clinical signs pointing to the presence of a syphilitic infection. The author suggests that an important subgroup of rheumatic children can be defined in whom the rheumatic infection is superimposed upon congenital syphilitic infection and untreated, undetected syphilis.

Barr, Sir James: The Preservation of a Healthy and Efficient Circulatory System From Childhood to Advanced Age. Brit. M. J. **1:** 769, 1930.

The author has interested himself in the physical side of the function of the heart and the peripheral circulation. He believes that through regulation of exercise and other influences, such as internal gland secretion, and elimination over long periods of years, that the circulation can be improved and altered. He stresses particularly abuse of the body from infection, alcohol, tobacco and exercise. The viewpoint expressed in this survey is extremely important and one which at times may be lost sight of.

Herpath, C. E. K., and Perry, C. B.: The Coronary Arteries in a Case of Familial Liability to Sudden Death. Brit. M. J. Apr. 12, 1930, p. 685.

This report supplements one made by Coombs and Lucas describing a family of whom the father and two sons had died with great suddenness. They had examined the heart of one of these sons, a man aged 32, who had been taken ill while playing football and had died within an hour or so. It was found to present macroscopical changes apparently of an atheromatous type in the coronary arteries.

The present report describes the findings in a third son who died suddenly. Electrocardiograms and the history and physical examination are given. Postmortem examination including radiograms of the injected heart specimens showed degenerative changes in the coronary artery. The authors describe the pathological process as one of premature senility of the arterial tree with a particular incidence on the coronary arteries.

Robey, William H., and Finland, Maxwell: Effect of Tonsillectomy on the Acute Attack of Rheumatic Fever. Arch. Int. Med. **45:** 772, 1930.

The authors present briefly some of the results of their experiments with enucleating the tonsils during the acute attack of rheumatic fever. In a period of five years there were 165 patients included in the study. Of these, 71 were operated on during their residence in the Boston City Hospital for polyarthritis, leaving 94 as controls. Among the 71 operative cases, there was definite clinical evidence of activity at the time of the operation in 50; the remaining 21 cases were apparently quiescent. Some of the patients had previously been subjected to tonsillectomy but about one-half of these had remains of tonsillar tissue.

The authors believe that the earlier the focus of infection is discovered, the greater the possibility of removing it, thus lessening the recurrence of attack, the length of time in the hospital and the danger of cardiac involvement. The

removal of the focus can be performed as readily at the end of one week as four. They believe that tonsillectomy may be performed during the active stage of acute rheumatic fever without harm to the patient from the operation. Tonsillectomy at this time offers no more dangers than when performed under what appear to be the most favorable conditions. They state that tonsillectomy may produce an exacerbation of the joint symptoms but that it will probably be mild and brief if the operation is a success. This point should be explained to patients beforehand.

Cobe, Herbert Marshall: The Incidence of Bacteria in 400 Tonsil Cultures. J. Infect. Dis. **46:** 298, 1930.

In 400 tonsillar cultures staphylococci were the predominating organism. Streptococci followed the pneumococci in predominance, with the hemolytic streptococci the predominant members of the group. Three per cent of the nonhemolytic streptococci recovered are classed as streptococcus cardioarthritidis. The nature of the disease which led the patient to have tonsillectomy is not stated.

From this study there appears to be a definite relationship between the type of organism recovered from tonsillar cultures and the age of the patient, streptococci being more common in younger patients. There also seems to be a definite seasonal difference in the organisms recovered from tonsillar cultures: B. influenzae, B. mucosuscapsulatus, and the diphtheroids all being more prevalent in the spring; Micrococcus catarrhalis more prevalent in the fall.

Lewis, Sir Thomas: Early Signs of Cardiac Failure of the Congestive Type. Brit. M. J. **1:** 849, 1930.

The author believes that the question of the capacity of the heart for work is a most important one when a patient seeks advice and disease of the heart is suspected. The answer to this question should dominate both the prognosis and treatment. He discusses among the early signs of cardiac failure, breathlessness, increased venous congestion, visible and venous pulsation and enlargement of the liver.

The author believes that attempts to estimate cardiac output in terms of fluid volume, while important from a physiological standpoint have as yet found no application in clinical work and are unlikely to find such application for many years to come.

Breathlessness occurs as first main symptom of cardiac failure. This breathlessness should be measured not in terms of degree but in terms of effort which produces this symptom. When breathlessness is present, when the patient is at rest cardiac failure is well established and other symptoms make their appearance.

In order to measure venous pressure, the author employs the simple manometer connected to a wide needle with the blood within a vein. When the blood enters this manometer, the level at which it rests in comparison with the level of the manubrium sterni and the level of the right auricle indicates the degree of venous obstruction. It is possible to note the point at which peripheral veins especially in the neck and in the arm collapse when compared with the level of the manubrium sterni. This older method with certain restrictions is a most satisfactory method for estimating venous congestion.

The author also points out that the level at which the venous pulsation reaches its maximum intensity may be utilized as the level of venous pressure. The details of these observations are explained.

He describes the observations of venous pulsation in normal and in abnormal subjects with venous congestion. The veins can be seen to pulsate in the superficial veins of the neck, or can be felt over the larger and deeper vessels in the neck.

Enlargement of the liver occurs in a latter stage of venous congestion and is a valuable sign of cardiac failure.

Hift, Robert: On Treatment of Luetic Aortitis. Wien. klin. Wchnschr. **42:** 33, 1929.

The importance of the serum reaction is emphasized. This may be negative in well-established cases. Also a positive reaction may occur in a resting infection.

Schottmüller's classification is given: supravalvular, valvular, coronary and aneurysmal lues. Luetic valvular disease or aneurysms do not alter the prognosis sufficiently to change the intensity of the treatment. The severity of the findings are not directly proportional to the intensity of the disease. It is more important to pay attention to the general health of the patients, and to interference with the circulation, hypertension, arteriosclerosis, etc. Subjective symptoms often count more than objective ones, and form the best indication of the progress of the treatment.

These are divided into symptoms from the central nervous system, pains (angina), cardiac neurosis, circulatory insufficiency, and symptoms due to aneurysms.

Only the pathological lesions which have not reached the stage of scar formation are susceptible to treatment. The clearing of active lesions may be preceded by flare-ups (Jarisch-Herxheimer reactions). This is probably a chemo-reaction of the tissues. It may be produced not only by salvarsan, but also by bismuth and mercury.

The author discusses the sudden death in this disease, and shows that it has nothing to do with the treatment, as it frequently occurs in cases not treated.

The duration of life from the onset of symptoms seems to be longer in patients who have been treated.

The Herxheimer reaction may produce status anginosus or precipitate congestive failure. Therefore treatment should be started gradually.

In one case untoward symptoms occurred after mercury while the patient tolerated bismuth and salvarsan, but as a rule treatments should be begun with mercury.

The author emphasizes the personal and moral element in the treatment and also the proper medical treatment of such patients as show interference with their circulation; this latter may be an important preliminary to anti-luetic treatment.

He also emphasizes that with the improved diagnostic and therapeutic technic the prognosis of aneurysm is very much improved. It is also the general impression that with systematic anti-luetic treatment the prognosis of luetic aortitis is much improved, the improvement in symptoms proceeds independently of changes in the sero-reaction.

The author has never seen improvements in the x-ray pictures after anti-luetic treatment. On physical examination there is no change.

Finally, he discusses the relative value of the various drugs and is of practically the same opinion as are the leading authorities in this country.

Hurxthal, Lewis M.: Auricular Fibrillation in Patients With Goiter. Am. J. M. Sc. **179:** 507, 1930.

This report deals with 59 cases of postoperative paroxysmal auricular fibrillation and 55 cases of established auricular fibrillation treated in the Lahey Clinic. All the latter were at one time or another associated with hyperthyroidism while a few of the former followed operation for nontoxic goiters. Diagnosis was made chiefly by auscultation in the paroxysmal type while electrocardiographic tracings were used in practically all of the established group.

In most instances paroxysmal auricular fibrillation is seen following operation. It occurs frequently following removal of nontoxic goiters but more often after partial or subtotal thyroidectomy in patients with primary hyperthyroidism. It is frequently disturbing to the patient but rarely does it produce any alarming systemic

reaction. Its appearance, therefore, cannot be considered as a cardiac emergency. Treatment in most instances is indicated only for the comfort of the patient. The author believes that quinidine yields the best results in the treatment of this type of patient. It has been used only if the irregularity is distressing to the patient or if it persists after two or three days.

Approximately 10 per cent of the patients with hyperthyroidism show established auricular fibrillation. Over 90 per cent of those patients who have frank congestive heart failure associated with hyperthyroidism have this irregularity and conversely from 30 to 40 per cent of those having auricular fibrillation have a history of various degrees of this type of heart failure. Of 55 cases of this type of fibrillation, 7 stopped spontaneously and in 5 other cases quinidine was not successful. Thirty-four cases were successfully treated, 4 of which recurred. Of the 34 cases successfully treated, 30 were first given treatment after operation while 4 of these had been given quinidine successfully before thyroidectomy. Two of these recurred after the operation, and were then again successfully restored to normal rhythm. Six patients received their treatment in periods of four months to four years after operation. Iodine and thyroidectomy caused cessation in 15 per cent of all operated cases during the period of hospital observation. Iodine, thyroidectomy and quinidine resulted in a permanent return to normal rhythm in 65 per cent of all operable cases, at least for the time in which they were under observation. In hyperthyroidism uncomplicated by cardiovascular disease, return to normal rhythm may be anticipated in 100 per cent of the cases.

The selection of cases with indications and contraindications of quinidine therapy is discussed.

Weiss, Soma: The Development of the Clinical Concept of Arterial Hypertension. New Eng. J. Med. **202:** 891, 1930.

This communication sketches the significant features of the evolution of the concept of hypertension and associated body changes. Emphasis is placed on the main features of this development rather than on the details of its history.

Stewart, Harold J.: The Use of Theocalcin in the Treatment of Heart Failure of the Congestive Type. J. Clin. Investigation. **8:** 389, 1930.

The author has studied the effect of administration of theocalcin (theobromine-calcium salicylate) to 16 patients suffering from heart failure of the congestive type while in the hospital. The usual precautions were taken to insure standard conditions during the period of observation. In one patient nausea and vomiting seemed to indicate a toxic effect. In another case it was not possible to separate the effect of theocalcin from the simultaneous action of digitalis and urea. It was administered to one patient without signs of congestion to ascertain its effect in the absence of edema and in this patient there was no diuresis. In a second patient exhibiting edema and ascites of nephritic origin, no diuresis resulted from the administration of the drug.

Satisfactory diuresis occurred in 11 of the remaining patients. In eight, the diuresis was sufficient to free the patient of signs of heart failure and in two it was partially effective, in one it was impossible to estimate the part it played. Most of these patients suffered from arteriosclerotic form of heart disease.

While no study was undertaken to determine the action of theocalcin it is presumed that the effect is directly on the kidney. The drug can be given as long as the diuresis lasts and as long as it maintains the output of urine near the fluid intake.

The author believes that theocalcin was by far the most effective diuretic administered during this study.